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Attorneys for Plaintiff
CALIFORNIA RESTAURANT ASSOCIATION

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

OAKLAND DIVISION

CALIFORNIA RESTAURANT ASSOCIATION,

Plaintiff,

v.

THE CITY AND COUNTY OF SAN FRANCISCO, et al.,

Defendants.

CALIFORNIA RESTAURANT ASSOCIATION,

Plaintiff,

v.

THE COUNTY OF SANTA CLARA, et al.,

Defendants.

Case No. CV-08-03247 (CW), and
Case No. CV-08-03685 (CW)

APPENDIX IN SUPPORT OF PLAINTIFF'S REPLY MEMORANDUM

Date: August 28, 2008
Time: 2:00 P.M.
Dept: Courtroom 2, 4th Floor
Judge: The Honorable Claudia Wilken

Complaints filed: July 3 and July 22, 2008, respectively

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Dated: August 22, 2008

ARNOLD & PORTER LLP

By: /s/
Trenton H. Norris
Attorneys for Plaintiff
CALIFORNIA RESTAURANT
ASSOCIATION

Exhibit A

Office of the City Attorney

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News Release: 2008-07-07

Herrera Blasts Secrecy by Fat-Peddling Chains, As Fast Food Lobby Sues Over Menu Labeling

Lawyers for Fast Food Chain Restaurants Assert First Amendment Right to Withhold Nutrition Information from Consumers on Menus

SAN FRANCISCO (July 7, 2008) -- City Attorney Dennis Herrera vowed a lean, aggressive defense to a lawsuit filed in Federal Court late last week that seeks to invalidate a San Francisco menu labeling ordinance requiring certain chain restaurants to disclose on their menus the amount of calories, saturated fat, carbohydrates and sodium for each listed item.

Representing chains including McDonald's and Burger King, the California Restaurant Association's civil complaint and related filings -- which at 59 total pages are nearly as bloated as Burger King's Triple Whopper Sandwich with Cheese (1,230 calories, 82 grams of fat) -- were filed in U.S. District Court for the Northern District of California in San Francisco late Thursday, July 3. The complaint obtained by City officials today alleges that San Francisco's ordinance violates both a federal statute governing nutrition disclosure standards and the U.S. Constitution's First Amendment, by compelling speech with which its member restaurants may disagree -- arguments Herrera likened to the nutritional merit of a McDonald's Chocolate Triple Thick 32 ounce shake (1,160 calories, 27 grams of fat).

In addition to a Court declaration that San Francisco's menu labeling ordinance is unconstitutional, the CRA is seeking blockage of the measure's implementation in a manner not unlike the kind of arterial obstructions that would likely result from frequent meals of a Quizno's footlong Tuna Melt (2,090 calories, 175 grams of fat). But a similar effort by CRA's New York State counterpart failed. In April, U.S. District Court Judge Richard J. Holwell held that New York City's menu labeling regulation is not pre-empted by federal law, and that "the required disclosure of caloric information is reasonably related to the government's interest in providing consumers with accurate nutritional information and therefore does not unduly infringe on the First Amendment rights" of affected chain restaurants.

"I think it's outrageous that fat-peddling chain restaurants are asserting a First Amendment right to keep consumers uninformed about the nutritional contents of their menu items," said Herrera, calling the irony as rich as an order of Outback Steakhouse Aussie Cheese Fries with Ranch Dressing (2,900 calories, 182 grams of fat). "At a time when public health departments are grappling with the grievous consequences of obesity, diabetes, and other serious health problems, cities like San Francisco are

justified to take steps that enable residents to make healthier choices. The City's menu labeling ordinance does not ban or restrict a single food item -- it is simply requires that consumers have better data to make informed choices."

San Francisco's ordinance was passed with unanimous support of the San Francisco Board of Supervisors on March 18, 2008, and signed into law by Mayor Gavin Newsom on March 24, 2008. Notably, the Golden Gate Restaurant Association, which represents San Francisco Bay Area restaurants, did not oppose the measure. The law amends the San Francisco Health Code to require chain restaurants to disclose, per standard menu item, the total number of calories on menu boards and food tags; to revise existing requirements for providing nutritional information on alternative disclosure media; and to authorize the Department of Public Health to impose a fee on chain restaurants for implementation of the ordinance.

Public opinion polls have shown consistent support for menu labeling requirements like those in New York City and San Francisco, with favorable responses nowhere more pronounced than in California. A 2007 study by the California Center for Public Health Advocacy found 84 percent of respondents in support of "requiring fast-food and chain restaurants to post nutrition information on their menus."

Copies of the CRA's complaint and related documents are available on the City Attorney's Web site at the following URL:

http://www.sfgov.org/cityattorney/

The case is California Restaurant Association v. City and County of San Francisco et al, U.S. District Court for the Northern District of California, Case No. 08-3247.

# # #

Exhibit B

RESEARCH BRIEF

Helping Consumers Make More Healthful Food Choices: Consumer Views on Modifying Food Labels and Providing Point-of-Purchase Nutrition Information at Quick-service Restaurants

Amy M. Lando, MPP; Judith Labiner-Wolfe, PhD

ABSTRACT

Objectives: To understand consumer (1) interest in nutrition information on food labels and quick-service restaurant menu boards and (2) reactions to modifying this information to help highlight calories and more healthful choices.

Design: Eight consumer focus groups, using a guide and stimuli.

Setting: Focus group discussions in 4 US cities.

Participants: A total of 68 consumers, with 7 to 10 per focus group.

Analysis: Authors prepared detailed summaries of discussions based on observation. Video recordings and transcripts were used to cross-check summaries. Data were systematically reviewed, synthesized, and analyzed.

Phenomenon of Interest: Consumer views on alternative presentations of nutrition information on packaged food items and quick-service restaurant menu boards.

Results: Participants (1) were interested in having nutrition information available, but would not use it at every eating occasion; (2) thought that food products typically consumed at 1 eating occasion should be labeled as a single serving; and (3) indicated that an icon on labels and menu boards that signaled more healthful options could be helpful.

Conclusions and Implications: Findings provide a basis for the development of more systematic studies to better understand whether alternative presentations of nutrition information would help consumers.

Key Words: nutrition policy, food labeling, food industry, focus groups

(J Nutr Educ Behav. 2007;39:157-163)

INTRODUCTION

The alarming increase in the prevalence of obesity in the United States has propelled this issue into the public health forefront. Associated with such health problems as diabetes, high blood pressure, asthma, arthritis, and elevated cholesterol, obesity threatens the health status of 44.3 million American adults.[1] The diversity of factors that have fed America's weight gain and allowed it to grow so substantially suggest that there is no one means to reverse course. Rather, there is a need to redress overweight and obesity from a multifaceted perspective that involves the many sources and exacerbating circumstances that contribute to calorie imbalance.

In 2003, US Food and Drug Administration (FDA) Commissioner Mark McClellen established the internal Obesity Working Group to study the obesity epidemic and to identify responsive strategies within the agency's purview. The immediate outcome of this initiative was a report outlining steps the agency would take to help fight obesity. This action plan covers several areas under FDA's regulatory authority, including exploring changes to labels on packaged food items. It also urges the restaurant industry to voluntarily offer point-of-purchase nutrition information to consumers.[2]

To help inform the Obesity Working Group's recommendations, the FDA conducted 8 consumer focus groups.

US Food and Drug Administration, Center for Food Safety and Applied Nutrition, College Park, Maryland.

This research was exclusively funded by the US Department of Health and Human Services.

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The focus groups were used to help enhance the agency's understanding of consumer interest in nutrition information on food labels and quick-service menu boards and reactions to alternative presentations of this information to help highlight calories and more healthful choices.

On food packages, FDA was specifically interested in consumer reaction to highlighting calories and other nutrition information on packages that contain more than 1 serving but are often consumed at a single eating occasion and front panel symbols that could be used as a signal for a healthful nutrition profile. On quick-service restaurant menu boards, FDA wanted to explore consumer reaction to placing calorie information beside each menu item, using a symbol to indicate more healthful meal combinations, and grouping more healthful meal combinations together. This paper describes the results of the focus groups.

STUDY DESCRIPTION

Study Design

In the Health Belief Model for behavior change, nutrition information on food labels and quick-service restaurant menu boards are potentially "cues to action" that play a role in helping consumers to make more healthful choices.[3] The focus groups were designed to explore consumer interest in and response to possible cues.

Focus groups have been shown to be an effective method for exploring the range of responses to alternative presentations of information.[4] The objective of these focus groups was to gain a preliminary, qualitative understanding of consumer interest and preferences. Although these focus groups cannot be generalized to the population, the results can help indicate a direction for future research.

Eight focus groups were conducted in 4 US cities: Calverton, Maryland; Bensalem, Pennsylvania; San Antonio, Texas; and Oak Brook, Illinois. The sites were chosen because they are geographically diverse from one another and in large population centers. Calverton, Maryland was specifically selected for its proximity to the FDA headquarters.

Participants

Purposive sampling was used to recruit participants from lists maintained by the focus group companies in each city. To qualify, participants had to be at least 18 years old. To help ensure that all participants could be actively involved in the discussion, participants also had to be regular grocery shoppers and have bought food at a quick-service restaurant in the past month. A screening questionnaire was developed and administered by phone to determine whether potential participants met these criteria and could be invited to a focus group.

The groups were segregated by education, separating those who had attended a 4-year college and those who had not. In each city, there was 1 higher and 1 lower education group. Segregating by education was used to help make the discussion accessible to all participants within a group. The groups were also segregated by gender, with 1 female group and 1 male group in each city. This segregation was done to ensure that participants felt comfortable speaking candidly about diet and weight issues.

Of the 8 focus groups, 2 were all female and higher education, 2 were all female and lower education, 2 were all male and higher education, and 2 were all male and lower education. There were 7 to 10 participants in each group and a total of 68 participants (Table).

Stimuli

Mock food labels and restaurant menu boards were designed to look similar to those consumers see regularly on packaged food items and at quick-service restaurants. To help design the labels and menu boards, the researchers studied options available in the market at the time.

The mock food labels were for a large, individually wrapped muffin (114 g), a 20-ounce plastic bottle of soda, and a family-sized frozen lasagna (1000 g). The muffin and soda were specifically chosen because they contained more than 1 serving per package (2 servings for the muffin and 2½ servings for the soda), but they are the type of products that are often consumed at 1 eating occasion. The lasagna was chosen to represent a meal

Table 1. Location, Education Level, Gender, and Number of Participants in Each of the 8 Focus Groups

	Location	Education Level	Gender	Number of Participants
Group I	Calverton, MD	Lower education	Female	8
Group II	Calverton, MD	Higher education	Male	10
Group III	Bensalem, PA	Higher education	Female	8
Group IV	Bensalem, PA	Lower education	Male	9
Group V	San Antonio, TX	Lower education	Female	9
Group VI	San Antonio, TX	Higher education	Male	9
Group VII	Oak Brook, IL	Higher education	Female	8
Group VIII	Oak Brook, IL	Lower education	Male	7

item. The menu boards were for a fictitious quick-service burger restaurant. Stimuli were modified slightly over the course of the study. Stimuli that tested poorly were revised as part of an iterative process.

Focus Group Discussions

All of the focus groups were conducted by an experienced moderator. The moderator's guide (Figure 1) was designed to meet the specific informational goals of the FDA's Obesity Working Group to understand what nutrition information is meaningful to consumers and how they react to food package labeling and menu boards designed to help them make more healthful choices.

After an initial icebreaker, respondents were asked how they make food choices and what factors most influence their purchase decisions. To mitigate any effects related to the order of topics, half of the groups initially discussed food labels, and the other half talked first about quick-service restaurants. Participants in the groups that started with food labels were asked to talk about packaged food products, including how they determine whether a packaged food product is healthful and whether they look for nutrition information on the label. They were also asked what nutrition information they look for when purchasing packaged food products and how frequently they look for such information.

1. Opening Remarks: Introduction, Rules, and Ice Breaker
2. Introduction: Food Choices
   - What are the most important factors that help you choose what foods to buy?
3. Food Labels General Discussion*
   - How do you determine whether a particular packaged food is nutritious?
   - How do you use the information on the Nutrition Facts Panel (NFP)?
   - When do you usually use the NFP? Do you use the NFP before or after you decide to purchase a food?
   - Once you have a product at home do you look at the NFP? When you look at it?
4. Baseline Labels (pass out 20 ounce soda and large muffin)
   - Overall impression of products
   - Do they seem high or low in calories?
   - Importance of calories in deciding to eat a food.
   - Discussion of the serving size of these products. Do you compare serving size to what you actually eat?
   - Discussion about percent daily value (%DV)
5. Food Label Modifications
   - Overall impression of the each label
   - Ask about specific changes to the label
     i. Are they useful for you? Why or why not?
6. Food Label Modification Summary
   - Which of these ideas do you like the best? Why?
   - Which do you like the least and why?
7. Nutrition Information at Quick-service Restaurant General Discussion*
   - Role of nutrition information when dining at fast food restaurants
   - Where would you like to see nutrition information in restaurants?
   - Currently, how do you obtain nutritional information for foods consumed away from home?
8. Baseline Quick-service Restaurant Menus
   - Overall impression of menu
   - What would you eat at this restaurant and why?
   - How do you determine the healthfulness of the food here?
9. Testing of Quick-service Restaurant Menu Boards
   - Overall impression of each menu board
   - What items would you eat on this menu? Why?
   - What do you like and dislike about each option?
10. Quick-service Menu Summary
    - Which of these ideas do you like the best? Why?
    - Which do you like the least and why?

*Half of the groups started with restaurants menus, the other half started with the food labels.

Figure 1. Outline of moderator's guide.

Following this discussion, participants reacted to a series of mock food labels for a large, clear-wrapped muffin and a 20-ounce plastic bottle of soda. The Nutrition Facts Panel information on the baseline mock labels was comparable to that found on muffin and soda products in the market and was consistent with FDA regulations.

Conversation on the baseline labels was followed by discussion of several modified labels. The modifications included: (1) substituting the "calories from fat" declaration with a Percent Daily Value (%DV) for calories, increasing the relative font size for calories, and changing the way serving size is declared; (2) adding a second column in the Nutrition Facts Panel with calories and other nutrition information for the entire package; and (3) declaring the number of calories on the front of the packages.

Participants also examined 2 versions of a lasagna package. One was for standard meat lasagna, and the other was for meat lasagna with a more healthful nutrition profile. The latter product displayed a keyhole symbol on the front of the package with a disclosure statement indicating that it met FDA's requirements for fat, saturated fat, cholesterol, and sodium to be labeled "healthy."[5] The idea for using a keyhole symbol to designate the product as "healthy" came from Sweden's use of a green keyhole symbol for food items that meet fat and fiber requirements.[6]

After the discussion about food labels concluded, the moderator shifted the respondents' attention to the topic of quick-service restaurants. The participants were asked how they make decisions when purchasing meals in quick-service venues.

Participants were asked to look at a series of mock quick-service restaurant menu boards. Each menu board offered a unique presentation of point-of-purchase information on calories and other nutrients. Modifications to the menu boards included: (1) printing calories next to each item; (2) grouping more healthful meal combinations together; and (3) identifying more healthful meal combinations with a keyhole symbol. To define more healthful meals, the following statement was included on the menu: "*Healthier Meals have 1/3 or less of a day's calories in a 2000 calorie diet. Also 1/3 or less of the Daily Value for saturated fat and cholesterol, less than 1/2 of the Daily Value for sodium and 7 grams or less of saturated plus trans fat.*"

The research protocol for this study was submitted to the FDA's Research Involving Human Subjects Committee (RIHSC). RIHSC exempted the study from review.

Analysis

The authors observed the groups and took extensive notes on the discussions. These notes consisted of impressions and verbatims. They used these notes to write a detailed summary for each pair of groups in each city. Each summary was structured by the discussion topics in the moderator's

guide to facilitate comparative analysis of the findings across cities. Videos and transcripts were used to cross-check the authors' notes and summaries and reconcile any discrepancies. The summaries were systematically analyzed and reviewed for common reactions, attitudes, and beliefs. To assess external validity of the findings, the authors reviewed their notes and summaries with at least 1 non-note–taking focus group observer.

FOCUS GROUP FINDINGS

Participant Interest In Nutrition Information

Participants expressed some interest in nutrition. Many said that they considered the healthfulness of food products before buying them. Some participants could easily name food items, such as salads, that they considered healthful and food items that they considered unhealthful, such as french fries. They could characterize these food items as healthful or unhealthful without looking for nutrition information.

Most participants were interested in having nutrition information available to them when they eat at quick-service restaurants. For packaged food items, some participants had rules of thumb for the amounts of calories, fat, carbohydrates and other nutrients that would be acceptable to them, and they used the Nutrition Facts Panel to find this information. Participants were more conscious of some nutrients than others, depending on their diet, their eating strategy, or health issues. As one participant explained, "I just try to keep my sugar content down. That's my main thing."

In general, participants said that they would not always take nutrition information into consideration when deciding what to eat. Taste, convenience, price, cravings, and their family's preferences were described as often at odds with healthful eating.

How Participants Reacted To Labeling Modifications

Food labels. Participants generally agreed that the muffin and soda were not healthful food items. Many said that they do not usually eat these products because they are high in calories, carbohydrates, and, in the case of the muffin, fat. Although the muffin was labeled as 2 servings and the soda as 2½, most of the participants thought that they would consume them at 1 eating occasion. They felt that these products should be labeled as single-serving products. A participant expressed the frustration heard from many: "I think the serving size of a half a muffin is ridiculous. You're going to eat the whole muffin."

Some participants knew to multiply by the number of servings to calculate calorie and nutrient values for the package. Others, however, were confused or made mathematical mistakes when trying to make these calculations. Regardless of their mathematical skills, many participants stated that calculating calories and nutrients on food labels was neither an interest nor a priority. Similar to a number of other participants, one said: "I don't want to have to go in a store and do mathematics."

The first modified package label participants saw substituted a %DV for calories in place of "calories from fat," enlarged the calorie information, and changed the way serving size was declared. "Serving size: ½ muffin" was changed to "Amount per ½ muffin serving." The new wording to describe the serving size was intended to make more explicit that the calories and nutrient values were for only part of the package. In general, the participants did not notice these changes. When the new wording for serving size was pointed out, most did not think it was an improvement over the existing language.

The second modified label had 2 %DV columns on the Nutrition Facts Panel, the first for a single serving (for example, ½ muffin), and the second for the entire package (for example, 1 muffin). In the first 4 groups, quantities of nutrients were only listed for one serving. Most of these participants said that this method made it difficult for them to determine, for example, whether the 11 grams of fat in the muffin were for one serving or the whole muffin.

After comments from these groups indicating that the benefits of more information outweighed the costs of a more text-heavy Nutrition Facts Panel, the product label was modified to include absolute amounts for both a single serving and the entire package (Figure 2). Participant re-

Nutrition Facts

Amount per 1/2 muffin (57g)
Servings Per Container 2

Amount	1/2 Muffin	1 Muffin
Calories	**210**	**420**
		% Daily Value*
Calories	11%	22%
Total Fat 11g, 22g	17%	34%
Saturated Fat 3g, 6g	15%	30%
Trans Fat 4g, 8g		
Cholesterol 40mg, 80mg	13%	27%
Sodium 200mg, 400mg	8%	16%
Total Carbohydrate 24g, 48g	8%	16%
Dietary Fiber 1g, 2g	4%	8%
Sugars 17g, 34g		
Protein 3g, 6g		
Vitamin A	0%	0%
Vitamin C	4%	8%
Calcium	2%	4%
Iron	4%	8%

*Percent Daily Values are based on a 2,000 calorie

Figure 2. Modified muffin label with two %DV columns. The first column is for a single serving (½ muffin), and the second column is for the entire package (1 muffin). In addition, calories are in a large font, and a %DV for calories replaced "calories from fat."

action to this modification was positive. However, some thought it was not necessary to list the amount for a single serving on these products. Nutrition information for the entire package was adequate for those who would consume the muffin or soda on 1 eating occasion. Some participants suggested removing the %DV column and listing only the absolute calories and nutrient values for a serving size and the whole package.

When discussing the %DV, participants noted that they do not use this information the way it currently appears on food labels. Some indicated that they did not understand the meaning of %DV. Others did not believe that this percentage was relevant since they did not consider themselves to be on a 2000-calorie per day diet. A 2000-calorie diet is the basis for the %DVs on the Nutrition Facts Panel.[7] One participant explained why she did not use the %DV: "I don't even look at them anymore because I know they're based on this 2000 calorie daily diet and I don't want to eat 2000 calories a day."

The few participants who did use %DV believed that it was a good way to estimate how much of a particular nutrient they were eating. They also believed that the %DV helped them gauge a healthful diet.

Participants in the first 4 groups were shown food labels with a starburst that highlighted the calories per serving on the front display area. This concept was tested to better understand how consumers would react to calorie information that appears on the primary display panel. Typically, this information is found on the back or side panel of a product. Many participants felt that the attention-grabbing look of a starburst on the front panel implied the calorie information was for the entire package, not just a serving.

The last 4 groups saw a more neutral-looking white square with calories for the entire package on the front panel. Reactions to this stimulus were mixed and relatively unenthusiastic. Some participants thought it could be helpful to have calories highlighted on the front of the package so that they would not have to look for the Nutrition Facts Panel. Others, however, thought calorie information on the front was not enough for them to make an informed choice. Many participants were accustomed to using the Nutrition Facts Panel and did not see the value of duplicate information on the front label.

An icon that represented a healthful choice was tested on the front panel of a frozen lasagna label. Half of the groups saw a lasagna label with a purple keyhole symbol directly above the statement "Meets FDA Healthy Meal Guidelines." Participants' reactions were generally favorable toward the symbol. They thought it could help them to select more healthful products without adding time to their grocery shopping. Some participants mentioned that they would look for the keyhole when they were in a hurry in the grocery store and did not have time to read and evaluate the Nutrition Facts Panel information. The participants thought this icon would be valuable if they understood the definition of the symbol and could trust that the symbol was used only on products that met a standard. They expressed some concern that these more healthful choices would cost more or that they would not taste as good.

Restaurant menu boards. Participants suggested many possible ways that quick-service restaurants could provide point-of-purchase nutrition information. Their suggestions included using food wrappers, tray liners, brochures, take-away bags, posters near the cashier, and menu boards.

Participants reacted to multiple versions of a menu board designed to look like one found at a typical quick-service burger restaurant. One board listed calories after each menu item. Although participants were concerned with multiple macronutrients, many thought that having just calories listed was a useful shorthand signal about the healthfulness of the item. Some participants cautioned that adding information to already busy menu boards could be confusing.

Most participants reacted favorably to the idea of placing more healthful combination meals in a separate section of the menu board (Figure 3). Participants appreciated the ability to identify more healthful options with a quick glance. Those who tend to order à la carte preferred to have calories listed after each item, whereas those who usually order a combination meal preferred to have calories summed for the entire meal.

The favorable impressions of the purple keyhole icon to designate a more healthful choice on food labels carried over to quick-service restaurant menu boards. As discussed in relation to food labels, participants noted that they would find the symbol valuable if it were to have a uniform, understandable definition.

DISCUSSION

Several of the modifications to the nutrition information on food labels and options for providing nutrition information at quick-service restaurants could cue participants to make more healthful choices. Participants favored having nutrition information available, even if they would not always use it to make food choices. Participants believed that the packaged food items they typically consume in 1 eating occasion should be labeled as such. They also believed that icons to designate more healthful options on food labels and on quick-service menus could make choosing such options easier.

Under FDA regulations, most packaged food products are required to include nutrition facts information based on a serving size. Serving size is presented in a household measure appropriate to a given food (like cups, tablespoons, pieces, or other units) followed by the equivalent metric quantity in parentheses. Serving sizes were designed to reflect amounts customarily consumed per eating occasion. The data for these reference values is based on the 1977-1978 and 1987-1988 Nationwide Food Consumption Surveys.[8]

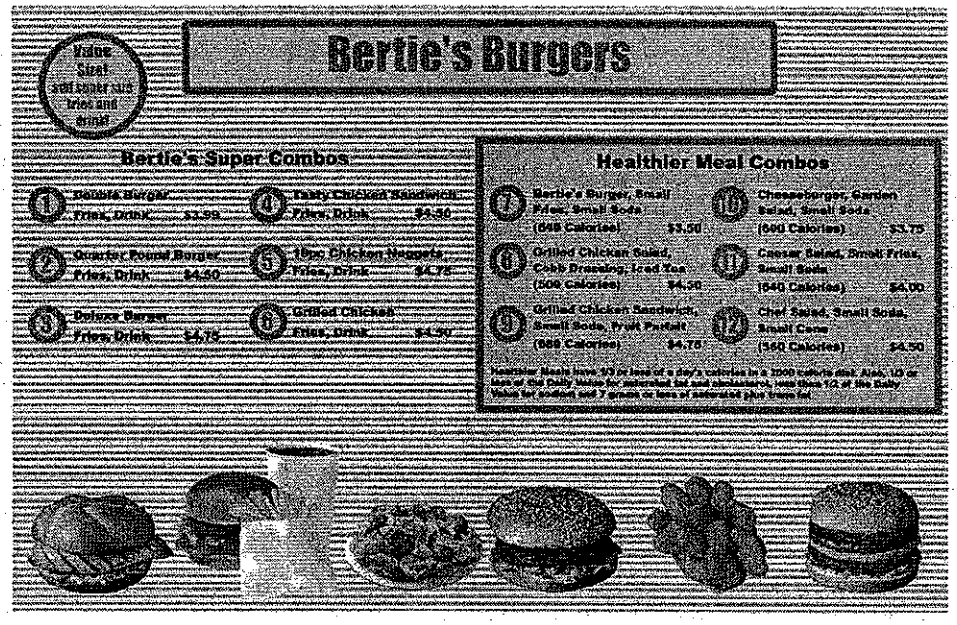

Figure 3. Fictitious restaurant menu with more healthful combination meals listed separately.

The basis of the serving size declaration on food labels may not be well aligned with current consumer behavior. Research suggests that portions consumed at one eating occasion have increased since these data were collected[9] and that consumers may underestimate what they consume when food typically consumed at 1 eating occasion is labeled for multiple servings.[10] Participants in the focus groups felt deceived when a quick glance at the Nutrition Facts Panel suggested fewer calories in the soda and less fat and fewer calories in the muffin than these products actually contained. Participants did not think they would do the calculations required to translate the information on serving size to the actual number of calories and amount of other nutrients they consumed. Some participants who attempted to multiply nutrients by the serving size made mistakes. By foregoing these calculations or miscalculating values, consumers may underestimate the number of calories and amounts of fat, cholesterol, sodium, and other nutrients consumed.

Major food companies such as Coca-Cola North America[11] and Kraft Foods, Inc.[12] have modified their labels on food products typically consumed in 1 eating occasion to provide nutrition information for the entire package.

Participants in the focus groups reacted favorably to the idea of having some type of nutrition information in quick-service restaurants, even though many said that the information might not always influence their meal selection. Food prepared away from the home is making up an increasing proportion of calories consumed by Americans.[13] Restaurants are not required to provide nutrition information, unless their food items are marketed with a health or nutrient content claim.[14] Consumers may have trouble judging the healthfulness of restaurant menu options in the absence of nutrition information.[15] However, studies have found that participants frequently avoided choosing the more healthful meal option when given nutrition information about that meal.[16,17] In a review of the literature on providing nutrition information to consumers in restaurants, Variyam concludes that, "the benefits of labeling (a change in consumer behavior leading to better nutritional outcomes) may be small or uncertain at best."[18]

Focus group participants generally reacted favorably to an icon designating healthful choices on food labels and at quick-service restaurants. They said that they would use the icon if its definition was understandable and trusted. Although information from the focus groups cannot determine whether consumers would actually use such a symbol if it were available, other countries have experimented successfully with a national symbol for food products that meet certain health requirements.[19] In the United States, individual companies and public health associations, including PepsiCo,[20] Kraft,[21] and The American Heart Association,[22] provide symbols to aid consumers in making more healthful choices.

Several of the food label modifications tested would not likely help participants make more healthful choices. Subtle changes to food labels went largely unnoticed. Many participants did not use or understand the current %DV column, and modifications did not improve its information value to participants.

Prior to issuing the current Nutrition Facts Panel label requirements, FDA conducted format research, which demonstrated that the %DV format could help consumers put information from the Nutrition Facts Panel into the context of the total daily diet.[23] Other research found that consumers may have difficulty un-

derstanding the meaning of %DV, even when they are able to use this information to rate packaged food products as low, medium, or high in fat.[24] Further research is needed on how to best convey nutrition information in the context of the daily diet.

Limitations

The sampling method for the groups was not probability based, and therefore, these focus group findings are not generalizable to a larger population. Another limitation of these focus group findings is that group dynamics may have influenced the information that participants decided to share and not to share. Despite these limitations, the groups do provide initial perspective on what cues might help consumers make more healthful choices.

IMPLICATIONS FOR RESEARCH AND PRACTICE

The focus group findings suggest that consumers are interested in using nutrition information on both food labels and at quick-service restaurants to select more healthful food items, at least some of the time. Focus group participants thought that food products typically consumed on 1 eating occasion should be labeled as single-serving. They thought that nutrition information should be readily available at quick-service restaurants. They also suggested that developing and branding an icon that signals more healthful products could help them to make better food choices. Finally, these findings suggest that further studies assessing how consumers would react to alternative presentations of nutrition information on food labels and on menu boards in quick-service restaurants may yield data that will prove valuable in improving cuing information that can help them maintain or improve their health and avoid overweight.

ACKNOWLEDGMENTS

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Exhibit C

Santa Clara County *Preliminary* List of Food Facilities that meet the Menu Labeling Ordinance

1. **AFC Sushi**
   459 Lagunita Drive, Stanford, CA 94305 (Stanford University)

2. **Burger King**
   261 Race Street, San Jose, CA 95126

3. **Jamba Juice**
   520 Lagunita Drive, Stanford, CA 94305 (Stanford University)

4. **Peet's Coffee & Tea**
   459 Lagunita Drive, Stanford, CA 94305 (Stanford University)

5. **Round Table Pizza**
   14940 Camden Avenue, San Jose, CA 95124

6. **Subway**
   459 Lagunita Drive, Stanford, CA 94305 (Stanford University)

7. **Taco Bell**
   1546 Camden Avenue, San Jose, CA 95008

8. **Togo's**
   14944 Camden Ave, San Jose, CA 95124

9. **USA Gas Station**
   1370 Camden Avenue, Campbell, CA 95008

Exhibit D

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Weight Loss with a Low-Carbohydrate, Mediterranean, or Low-Fat Diet

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ABSTRACT

BACKGROUND

Trials comparing the effectiveness and safety of weight-loss diets are frequently limited by short follow-up times and high dropout rates.

METHODS

In this 2-year trial, we randomly assigned 322 moderately obese subjects (mean age, 52 years; mean body-mass index [the weight in kilograms divided by the square of the height in meters], 31; male sex, 86%) to one of three diets: low-fat, restricted-calorie; Mediterranean, restricted-calorie; or low-carbohydrate, non–restricted-calorie.

RESULTS

The rate of adherence to a study diet was 95.4% at 1 year and 84.6% at 2 years. The Mediterranean-diet group consumed the largest amounts of dietary fiber and had the highest ratio of monounsaturated to saturated fat ($P<0.05$ for all comparisons among treatment groups). The low-carbohydrate group consumed the smallest amount of carbohydrates and the largest amounts of fat, protein, and cholesterol and had the highest percentage of participants with detectable urinary ketones ($P<0.05$ for all comparisons among treatment groups). The mean weight loss was 2.9 kg for the low-fat group, 4.4 kg for the Mediterranean-diet group, and 4.7 kg for the low-carbohydrate group ($P<0.001$ for the interaction between diet group and time); among the 272 participants who completed the intervention, the mean weight losses were 3.3 kg, 4.6 kg, and 5.5 kg, respectively. The relative reduction in the ratio of total cholesterol to high-density lipoprotein cholesterol was 20% in the low-carbohydrate group and 12% in the low-fat group ($P=0.01$). Among the 36 subjects with diabetes, changes in fasting plasma glucose and insulin levels were more favorable among those assigned to the Mediterranean diet than among those assigned to the low-fat diet ($P<0.001$ for the interaction among diabetes and Mediterranean diet and time with respect to fasting glucose levels).

CONCLUSIONS

Mediterranean and low-carbohydrate diets may be effective alternatives to low-fat diets. The more favorable effects on lipids (with the low-carbohydrate diet) and on glycemic control (with the Mediterranean diet) suggest that personal preferences and metabolic considerations might inform individualized tailoring of dietary interventions. (ClinicalTrials.gov number, NCT00160108.)

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THE DRAMATIC INCREASE IN OBESITY worldwide remains challenging and underscores the urgent need to test the effectiveness and safety of several widely used weight-loss diets.[1-3] Low-carbohydrate, high-protein, high-fat diets (referred to as low-carbohydrate diets) have been compared with low-fat, energy-restricted diets.[4-9] A meta-analysis of five trials with 447 participants[10] and a recent 1-year trial involving 311 obese women[4] suggested that a low-carbohydrate diet is a feasible alternative to a low-fat diet for producing weight loss and may have favorable metabolic effects. However, longer-term studies are lacking.[4,10] A Mediterranean diet with a moderate amount of fat and a high proportion of monounsaturated fat provides cardiovascular benefits.[11] A recent review citing several trials[12] included a few that suggested that the Mediterranean diet was beneficial for weight loss.[13,14] However, this positive effect has not been conclusively demonstrated.[15]

Common limitations of dietary trials include high attrition rates (15 to 50% within a year), small size, short duration, lack of assessment of adherence, and unequal intensity of intervention.[10,12,15-17] We conducted the 2-year Dietary Intervention Randomized Controlled Trial (DIRECT) to compare the effectiveness and safety of three nutritional protocols: a low-fat, restricted-calorie diet; a Mediterranean, restricted-calorie diet; and a low-carbohydrate, non–restricted-calorie diet.

METHODS

ELIGIBILITY AND STUDY DESIGN

We conducted the trial between July 2005 and June 2007 in Dimona, Israel, in a workplace at a research center with an on-site medical clinic. Recruitment began in December 2004. The criteria for eligibility were an age of 40 to 65 years and a body-mass index (BMI, the weight in kilograms divided by the square of the height in meters) of at least 27, or the presence of type 2 diabetes (according to the American Diabetes Association criteria[18]) or coronary heart disease, regardless of age and BMI. Persons were excluded if they were pregnant or lactating, had a serum creatinine level of 2 mg per deciliter (177 μmol per liter) or more, had liver dysfunction (an increase by a factor of at least 2 above the upper limit of normal in alanine aminotransferase and aspartate aminotransferase levels), had gastrointestinal problems that would prevent them from following any of the test diets, had active cancer, or were participating in another diet trial.

The participants were randomly assigned within strata of sex, age (below or above the median), BMI (below or above the median), history of coronary heart disease (yes or no), history of type 2 diabetes (yes or no), and current use of statins (none, <1 year, or ≥1 year) with the use of Monte Carlo simulations. The participants received no financial compensation or gifts. The study was approved and monitored by the human subjects committee of Soroka Medical Center and Ben-Gurion University. Each participant provided written informed consent.

The members of each of the three diet groups were assigned to subgroups of 17 to 19 participants, with six subgroups for each group. Each diet group was assigned a registered dietitian who led all six subgroups of that group. The dietitians met with their groups in weeks 1, 3, 5, and 7 and thereafter at 6-week intervals, for a total of 18 sessions of 90 minutes each. We adapted the Israeli version (developed by the Maccabi Health Maintenance Organization) of the diabetes-prevention program[19] and developed additional themes for each diet group (see Supplementary Appendix 1, available with the full text of this article at www.nejm.org). In order to maintain equal intensity of treatment, the workshop format and the quality of the materials were similar among the three diet groups, except for instructions and materials specific to each diet strategy. Six times during the 2-year intervention, another dietitian conducted 10-to-15-minute motivational telephone calls with participants who were having difficulty adhering to the diets and gave a summary of each call to the group dietitian. In addition, a group of spouses received education to strengthen their support of the participants (data not shown).

LOW-FAT DIET

The low-fat, restricted-calorie diet was based on American Heart Association[20] guidelines. We aimed at an energy intake of 1500 kcal per day for women and 1800 kcal per day for men, with 30% of calories from fat, 10% of calories from saturated fat, and an intake of 300 mg of cholesterol per day. The participants were counseled to consume low-fat grains, vegetables, fruits, and legumes and to limit their consumption of additional fats, sweets, and high-fat snacks.

MEDITERRANEAN DIET

The moderate-fat, restricted-calorie, Mediterranean diet was rich in vegetables and low in red meat, with poultry and fish replacing beef and lamb. We restricted energy intake to 1500 kcal per day for women and 1800 kcal per day for men, with a goal of no more than 35% of calories from fat; the main sources of added fat were 30 to 45 g of olive oil and a handful of nuts (five to seven nuts, <20 g) per day. The diet is based on the recommendations of Willett and Skerrett.[21]

LOW-CARBOHYDRATE DIET

The low-carbohydrate, non–restricted-calorie diet aimed to provide 20 g of carbohydrates per day for the 2-month induction phase and immediately after religious holidays, with a gradual increase to a maximum of 120 g per day to maintain the weight loss. The intakes of total calories, protein, and fat were not limited. However, the participants were counseled to choose vegetarian sources of fat and protein and to avoid trans fat. The diet was based on the Atkins diet (see Supplementary Appendix 2).[22]

NUTRITIONAL AND COLOR LABELING OF FOOD IN THE CAFETERIA

Lunch is typically the main meal in Israel. The self-service cafeteria in the workplace provided a varied menu and was the exclusive source of lunch for the participants. A dietitian worked closely with the kitchen staff to adjust specific food items to specific diet groups. Each food item was provided with a label showing the number of calories and the number of grams of carbohydrates, fat, and saturated fat, according to an analysis based on the Israeli nutritional database. Each food item was also labeled with a full circle (indicating "feel free to consume") or a half circle (indicating "consume in moderation"). The labels were color-coded according to diet group and were updated daily (see Supplementary Appendix 2).[23]

ELECTRONIC QUESTIONNAIRES AT BASELINE AND FOLLOW-UP

Adherence to the diets was evaluated by a validated food-frequency questionnaire[24] that included 127 food items and three portion-size pictures for 17 items.[25] A subgroup of participants completed two repeated 24-hour dietary recalls to verify absolute intake (data not shown). We used a validated questionnaire to assess physical activity.[26] At baseline and at 6, 12, and 24 months of follow-up, the questionnaires were self-administered electronically through the workplace intranet. The 15% of participants who requested aid in completing the questionnaires were assisted by the study nurse. The electronic questionnaire helped to ensure completeness of the data by prompting the participant when a question was not answered, and it permitted rapid automated reporting by the group dietitians.

OUTCOMES

The participants were weighed without shoes to the nearest 0.1 kg every month. With the use of a wall-mounted stadiometer, height was measured to the nearest millimeter at baseline for determination of BMI. Waist circumference was measured halfway between the last rib and the iliac crest. Blood pressure was measured every 3 months with the use of an automated system (Datascop Acutor 4) after 5 minutes of rest.

Blood samples were obtained by venipuncture at 8 a.m. after a 12-hour fast at baseline and at 6, 12, and 24 months and were stored at −80°C until an assay for lipids, inflammatory biomarkers, and insulin could be performed. Levels of fasting plasma glucose, glycated hemoglobin, and liver enzymes were measured in fresh samples. The level of glycated hemoglobin was determined with the use of Cobas Integra reagents and equipment. Serum levels of total cholesterol, high-density-lipoprotein (HDL) cholesterol, low-density-lipoprotein (LDL) cholesterol, and triglycerides were determined enzymatically with a Wako R-30 automatic analyzer, with coefficients of variation of 1.3% for cholesterol and 2.1% for triglycerides. Plasma insulin levels were measured with the use of an enzyme immunometric assay (Immulite automated analyzer, Diagnostic Products), with a coefficient of variation of 2.5%. Plasma levels of high-molecular-weight adiponectin were measured by an enzyme-linked immunosorbent assay (ELISA) (AdipoGen or Axxora), with a coefficient of variation of 4.8%. Plasma leptin levels were assessed by ELISA (Mediagnost), with a coefficient of variation of 2.4%. Plasma levels of high-sensitivity C-reactive protein were measured by ELISA (DiaMed), with a coefficient of variation of 1.9%. The clinic and laboratory staff members were unaware of the treatment assignments, and the study coordinators were unaware of all outcome data until the end of the intervention.

STATISTICAL ANALYSIS

For weight loss, the prespecified primary aim was the change in weight from baseline to 24 months. We used the Israeli food database[23] in the analysis of the results of the dietary questionnaires. We analyzed the dietary-composition data and biomarkers with the use of raw unadjusted means, without imputation of missing data. We compared the dietary-intake values between groups at each time point with the use of an analysis of variance in which all pairwise comparisons among the three diet groups were performed with the use of Tukey's Studentized range test. We transformed physical-activity scores into metabolic equivalents per week[27] according to the amount of time spent in various forms of exercise per week, with each activity weighted in terms of its level of intensity. For intention-to-treat analyses, we included all 322 participants and used the most recent values for weight and blood pressure. To evaluate the repeated measurements over time, we used generalized estimating equations for panel data analysis, also known as cross-sectional time-series analysis, with the use of the Stata software XTGEE command; this allowed us to account for the nonindependence of repeated measurements of the same bioindicator in the same participant over time. We used age, sex, time point, and diet group as explanatory variables in our models. To study changes over time and the effects of sex or the presence or absence of diabetes, we added appropriate interaction terms. We assessed the within-person changes from baseline in each diet group with the use of pairwise comparisons. We calculated the homeostasis model assessment of insulin resistance (HOMA-IR) according to the following equation[28]: insulin (U/ml) × fasting glucose (mmol/liter) ÷ 22.5. For a mean (±SD) difference between groups of at least 2±10 kg of weight loss, with 100 participants per group and a type I error of 5%, the power to detect significant differences in weight loss is greater than 90%. We used SPSS software, version 15, and Stata software, version 9, for the statistical analysis.

RESULTS

CHARACTERISTICS OF THE PARTICIPANTS

The baseline characteristics of the participants are shown in Table 1. The mean age was 52 years and the mean BMI was 31. Most participants (86%) were men. The overall rate of adherence (Fig. 1) was 95.4% at 12 months and 84.6% at 24 months; the 24-month adherence rates were 90.4% in the low-fat group, 85.3% in the Mediterranean-diet group, and 78.0% in the low-carbohydrate group (P=0.04 for the comparison among diet groups). During the study, there was little change in usage of medications, and there were no significant differences among groups in the amount of change; four participants initiated and three stopped cholesterol-lowering therapy. Twenty participants initiated blood-pressure treatment, five initiated medications for glycemic control, and one reduced the dosage of medications for glycemic control.

DIETARY INTAKE, ENERGY EXPENDITURE, AND URINARY KETONES

At baseline, there were no significant differences in the composition of the diets consumed by participants assigned to the low-fat, Mediterranean, and low-carbohydrate diets. Daily energy intake, as assessed by the food-frequency questionnaire, decreased significantly at 6, 12, and 24 months in all diet groups as compared with baseline (P<0.001); there were no significant differences among the groups in the amount of decrease (Table 2). The low-carbohydrate group had a lower intake of carbohydrates (P<0.001) and higher intakes of protein (P<0.001), total fat (P<0.001), saturated fat (P<0.001), and total cholesterol (P=0.04) than the other groups. The Mediterranean-diet group had a higher ratio of monounsaturated to saturated fat than the other groups (P<0.001) and a higher intake of dietary fiber than the low-carbohydrate group (P=0.002). The low-fat group had a lower intake of saturated fat than the low-carbohydrate group (P=0.02). The amount of physical activity increased significantly from baseline in all groups, with no significant difference among groups in the amount of increase. The proportion of participants with detectable urinary ketones at 24 months was higher in the low-carbohydrate group (8.3%) than in the low-fat group (4.8%) or the Mediterranean-diet group (2.8%) (P=0.04).

WEIGHT LOSS

A phase of maximum weight loss occurred from 1 to 6 months and a maintenance phase from 7 to 24 months. All groups lost weight, but the reductions were greater in the low-carbohydrate and the Mediterranean-diet groups (P<0.001 for the inter-

Table 1. Baseline Characteristics of the Study Population.*

Characteristic	Low-Fat Diet (N=104)	Mediterranean Diet (N=109)	Low-Carbohydrate Diet (N=109)	All (N=322)
Age — yr	51±7	53±6	52±7	52±7
Male sex — no. (%)	89 (86)	89 (82)	99 (91)	277 (86)
Current smoker — no. (%)	19 (18)	16 (15)	16 (15)	51 (16)
Weight — kg	91.3±12.3	91.1±13.6	91.8±14.3	91.4±13.4
BMI	30.6±3.2	31.2±4.1	30.8±3.5	30.9±3.6
Blood pressure — mm Hg†				
Systolic	129.6±13.2	133.1±14.1	130.8±15.1	131.3±14.5
Diastolic	79.1±9.1	80.6±9.2	79.4±9.1	79.7±9.2
Waist circumference — cm‡	105.3±9.2	106.2±9.1	106.3±9.1	105.9±9.1
Type 2 diabetes — no. (%)	12 (12)	15 (14)	19 (17)	46 (14)
Coronary heart disease — no. (%)	38 (37)	46 (42)	34 (31)	118 (37)
Blood biomarkers				
Serum LDL cholesterol — mg/dl	117.0±35.6	122.8±34.4	117.2±34.5	119.0±34.8
Serum HDL cholesterol — mg/dl	38.6±9.6	39.4±9.4	37.5±8.7	38.5±9.2
Serum triglycerides — mg/dl	156.5±62.4	173.6±67.7	181.7±116.9	170.8±90.0
Serum non-HDL cholesterol — mg/dl	154.2±37.9	163.3±35.8	161.3±36.0	159.6±36.7
Ratio of total cholesterol to HDL cholesterol	5.2±1.4	5.4±1.6	5.6±1.7	5.4±1.6
Fasting plasma insulin — μU/ml	13.3±6.8	14.6±8.0	14.1±10.2	14.0±8.5
Fasting plasma glucose — mg/dl	86.9±26.0	94.3±38.1	92.6±28.5	91.3±31.5
HOMA-IR	2.9±1.8	3.6±2.9	3.2±2.9	3.2±2.6
Plasma high-sensitivity C-reactive protein — mg/liter	3.6±2.9	4.6±3.4	4.5±3.3	4.2±3.2
Plasma high-molecular-weight adiponectin — mg/dl	7.3±2.6	7.3±2.8	7.3±3.1	7.3±2.8
Plasma leptin — mg/dl	12.0±9.6	14.2±13.2	11.2±8.1	12.5±10.5
Plasma bilirubin — mg/dl	0.74±0.34	0.79±0.35	0.77±0.36	0.77±0.34
Plasma alkaline phosphatase — U/liter	71.4±18.0	74.2±20.1	71.6±17.7	72.4±18.6
Plasma alanine aminotransferase — U/liter	28.2±14.2	27.7±12.6	28.3±11.2	28.1±12.7
Medications in current use				
Lipid-lowering therapy — no. (%)	28 (27)	29 (27)	27 (25)	84 (26)
Antihypertensive therapy — no. (%)	23 (22)	37 (34)	36 (33)	96 (30)
Hormone-replacement therapy — no. (%)	0	3 (3)	4 (4)	7 (2)
Oral glycemic-control medications — no. (%)	6 (6)	7 (6)	13 (12)	26 (8)
Insulin treatment — no. (%)	2 (2)	0	2 (2)	4 (1)

* Plus–minus values are means ±SD. To convert values for cholesterol to millimoles per liter, multiply by 0.02586. To convert values for triglycerides to millimoles per liter, multiply by 0.01129. To convert values for glucose to millimoles per liter, multiply by 0.05551. To convert values for bilirubin to micromoles per liter, multiply by 17.1. BMI denotes body-mass index, HDL high-density lipoprotein, HOMA-IR homeostasis model assessment of insulin resistance, and LDL low-density lipoprotein.
† Data were available from 297 participants.
‡ Data were available from 302 participants.

action between diet group and time) than in the low-fat group (Fig. 2). The overall weight changes among the 322 participants at 24 months were −2.9±4.2 kg for the low-fat group, −4.4±6.0 kg for the Mediterranean-diet group, and −4.7±6.5 kg for the low-carbohydrate group. Among the 277 male participants, the mean 24-month weight changes were −3.4 kg (95% confidence interval [CI], −4.3 to −2.5) for the low-fat group, −4.0 kg (95% CI, −5.1 to −3.0) for the Mediterranean-diet

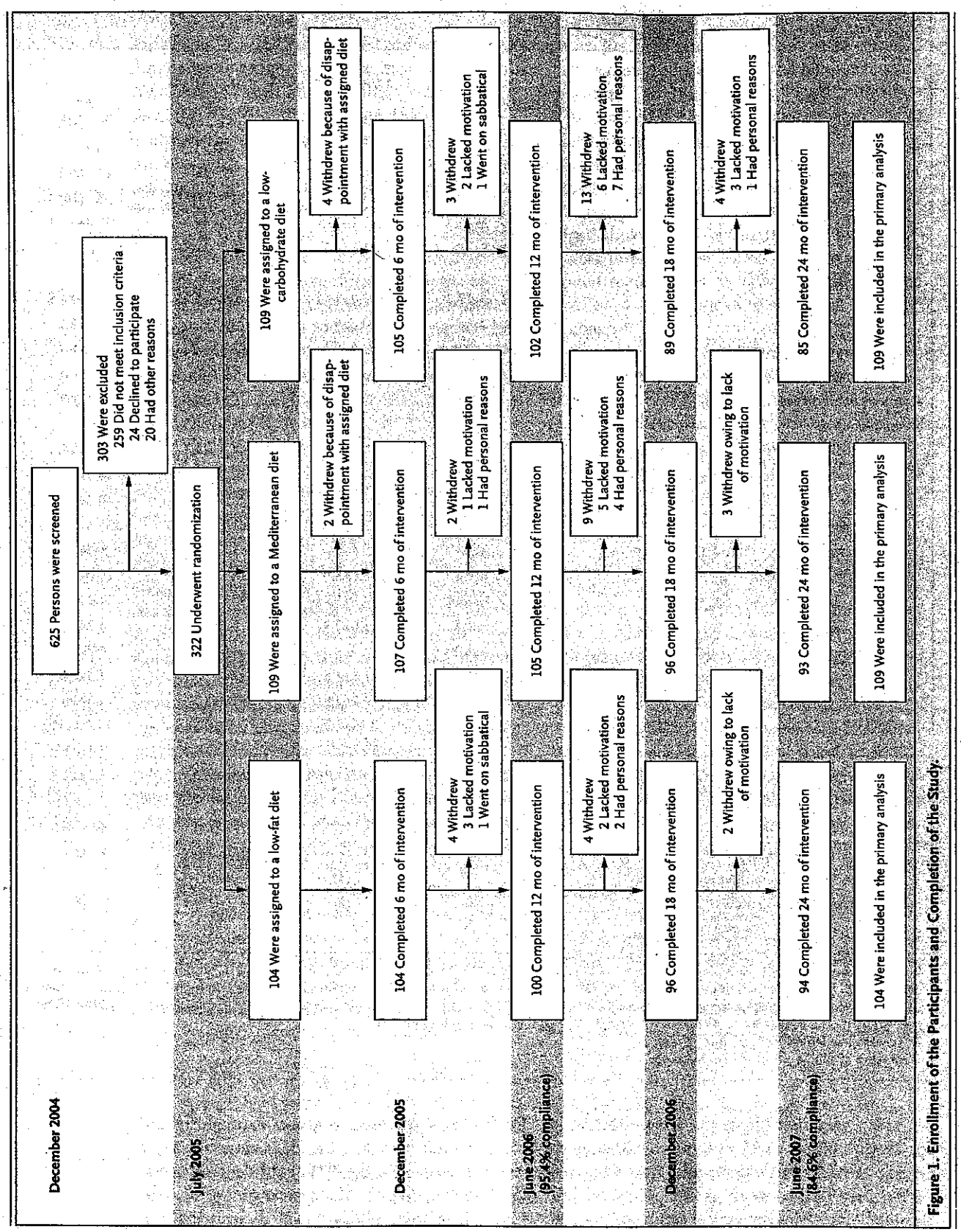

Figure 1. Enrollment of the Participants and Completion of the Study.

group, and −4.9 kg (95% CI, −6.2 to −3.6) for the low-carbohydrate group. Among the 45 women, the mean 24-month weight changes were −0.1 kg (95% CI, −2.2 to 1.9) for the low-fat group, −6.2 kg (95% CI, −10.2 to −1.9) for the Mediterranean-diet group, and −2.4 kg (95% CI, −6.9 to 2.2) for the low-carbohydrate group (P<0.001 for the interaction between diet group and sex). The mean weight changes among the 272 participants who completed 24 months of intervention were −3.3±4.1 kg in the low-fat group, −4.6±6.0 kg in the Mediterranean-diet group, and −5.5±7.0 kg in the low-carbohydrate group (P=0.03 for the comparison between the low-fat and the low-carbohydrate groups at 24 months). The mean (±SD) changes in BMI were −1.0±1.4 in the low-fat group, −1.5±2.2 in the Mediterranean-diet group, and −1.5±2.1 in the low-carbohydrate group (P=0.05 for the comparison among groups).

All groups had significant decreases in waist circumference and blood pressure, but the differences among the groups were not significant. The waist circumference decreased by a mean of 2.8±4.3 cm in the low-fat group, 3.5±5.1 cm in the Mediterranean-diet group, and 3.8±5.2 cm in the low-carbohydrate group (P=0.33 for the comparison among groups). Systolic blood pressure fell by 4.3±11.8 mm Hg in the low-fat group, 5.5±14.3 mm Hg in the Mediterranean-diet group, and 3.9±12.8 mm Hg in the low-carbohydrate group (P=0.64 for the comparison among groups). The corresponding decreases in diastolic pressure were 0.9±8.1, 2.2±9.5, and 0.8±8.7 mm Hg (P=0.43 for the comparison among groups).

LIPID PROFILES

Changes in lipid profiles during the weight-loss and maintenance phases are shown in Figure 3. HDL cholesterol (Fig. 3A) increased during the weight-loss and maintenance phases in all groups, with the greatest increase in the low-carbohydrate group (8.4 mg per deciliter [0.22 mmol per liter], P<0.01 for the interaction between diet group and time), as compared with the low-fat group (6.3 mg per deciliter [0.16 mmol per liter]). Triglyceride levels (Fig. 3B) decreased significantly in the low-carbohydrate group (23.7 mg per deciliter [0.27 mmol per liter], P=0.03 for the interaction between diet group and time), as compared with the low-fat group (2.7 mg per deciliter [0.03 mmol per liter]). LDL cholesterol levels (Fig. 3C) did not change significantly within groups, and there were no significant differences between the groups in the amount of change. Overall, the ratio of total cholesterol to HDL cholesterol (Fig. 3D) decreased during both the weight-loss and the maintenance phases. The low-carbohydrate group had the greatest improvement, with a relative decrease of 20% (P=0.01 for the interaction between diet group and time), as compared with a decrease of 12% in the low-fat group.

HIGH-SENSITIVITY C-REACTIVE PROTEIN, HIGH-MOLECULAR-WEIGHT ADIPONECTIN, AND LEPTIN

The level of high-sensitivity C-reactive protein decreased significantly (P<0.05) only in the Mediterranean-diet group (21%) and the low-carbohydrate group (29%), during both the weight-loss and the maintenance phases, with no significant differences among the groups in the amount of decrease (Fig. 4A). During both the weight-loss and the maintenance phases, the level of high-molecular-weight adiponectin (Fig. 4B) increased significantly (P<0.05) in all diet groups, with no significant differences among the groups in the amount of increase. Circulating leptin, which reflects body-fat mass, decreased significantly (P<0.05) in all diet groups, with no significant differences among the groups in the amount of decrease; the decrease in leptin paralleled the decrease in body weight during the two phases (Fig. 4C). The interaction between the effects of low-carbohydrate diet and sex on the reduction of leptin (P=0.04), as compared with the low-fat diet, reflects the greater effect of the low-carbohydrate diet among men.

FASTING PLASMA GLUCOSE, HOMA-IR, AND GLYCATED HEMOGLOBIN

Among the 36 participants with diabetes (Fig. 4D), only those in the Mediterranean-diet group had a decrease in fasting plasma glucose levels (32.8 mg per deciliter); this change was significantly different from the increase in plasma glucose levels among participants with diabetes in the low-fat group (P<0.001 for the interaction between diet group and time). There was no significant change in plasma glucose level among the participants without diabetes (P<0.001 for the interaction among diabetes and Mediterranean diet and time). In contrast, insulin levels (Fig. 4E) decreased significantly in participants with diabetes and in those without diabetes in all diet groups, with no significant differences among groups in the

amount of decrease. Among the participants with diabetes, the decrease in HOMA-IR at 24 months (Fig. 4F) was significantly greater in those assigned to the Mediterranean diet than in those assigned to the low-fat diet (2.3 and 0.3, respectively; P=0.02; P=0.04 for the interaction among diabetes and Mediterranean diet and time). Among the participants with diabetes, the proportion of glycated hemoglobin at 24 months decreased by 0.4±1.3% in the low-fat group, 0.5±1.1% in the Mediterranean-diet group, and 0.9±0.8% in the low-carbohydrate group. The changes were signifi-

Table 2. Changes in Dietary Intake, Energy Expenditure, and Urinary Ketones during 2 Years of Intervention.*

Variable	Low-Fat Diet	Mediterranean Diet	Low-Carbohydrate Diet	P Value†
Energy change from baseline (kcal/day)				
6 mo	−458.3±1412.9	−254.6±740.6	−560.8±1568.3	0.22
12 mo	−559.1±1764.8	−321.7±802.4	−591.1±1472.9	0.33
24 mo	−572.6±1638.0	−371.9±864.2	−550.0±1453.9	0.55
Carbohydrate				
% of energy				
Baseline	51.8±8.1	51.5±8.3	50.8±8.4	0.63
6 mo	50.4±6.9	49.8±8.0	41.4±9.3‡	<0.001
12 mo	50.5±6.8	50.0±7.7	41.6±8.8‡	<0.001
24 mo	50.7±5.7	50.2±8.6	40.4±7.1‡	<0.001
Change from baseline (g/day)				
6 mo	−69.1±191.7	−39.6±98.1	−123.6±223.5‡	0.003
12 mo	−83.4±215.8	−49.0±112.3	−127.7±216.3‡	0.01
24 mo	−82.8±202.0	−50.5±126.1	−129.8±212.6‡	0.02
Protein				
% of energy				
Baseline	18.2±3.3	18.4±3.8	18.7±3.8	0.61
6 mo	19.6±3.7	18.9±3.6	21.6±3.5‡	<0.001
12 mo	19.4±3.4	18.9±3.6	21.5±4.0‡	<0.001
24 mo	19.0±3.2	18.8±3.5	21.8±3.9‡	<0.001
Change from baseline (g/day)				
6 mo	−12.8±58.7	−10.2±47.7	−10.2±69.7	0.99
12 mo	−16.7±84.9	−12.2±47.6	−11.8±69.3	0.86
24 mo	−19.8±75.0	−17.5±48.1	−6.9±66.6	0.38
Total fat				
% of energy				
Baseline	31.4±4.7	31.7±4.9	32.1±5.5	0.65
6 mo	30.7±4.0	33.2±5.1	38.8±6.9‡	<0.001
12 mo	30.8±4.2	32.9±5.1	38.5±6.5‡	<0.001
24 mo	30.0±3.9	33.1±5.5	39.1±5.5¶	<0.001
Change from baseline (g/day)				
6 mo	−14.7±55.6	−5.8±30.8	−3.6±58.2	0.23
12 mo	−18.0±68.1	−8.3±33.1	−4.8±57.7	0.21
24 mo	−18.9±66.4	−10.5±33.2	−1.7±51.2	0.10

Table 2. (Continued.)

Variable	Low-Fat Diet	Mediterranean Diet	Low-Carbohydrate Diet	P Value†
Saturated fat				
% of energy				
Baseline	9.7±2.4	9.7±2.4	9.9±2.3	0.84
6 mo	9.6±1.9	9.7±2.5	12.2±3.9‡	<0.001
12 mo	9.8±2.1	9.6±2.1	12.5±3.8‡	<0.001
24 mo	9.6±1.8	9.6±2.2	12.3±3.2‡	<0.001
Change from baseline (g/day)				
6 mo	−4.8±17.0	−3.0±11.4	0.28±18.7	0.07
12 mo	−5.3±21.3	−3.8±11.5	0.78±19.5§	0.04
24 mo	−6.2±21.4	−4.6±11.3	0.56±15.7§	0.02
Change in monounsaturated:saturated fat ratio from baseline				
6 mo	0.00±0.18	0.09±0.21¶	−0.02±0.23	0.001
12 mo	−0.02±0.18	0.08±0.19¶	−0.05±0.22	<0.001
24 mo	0.02±0.21	0.11±0.20¶	−0.01±0.21	<0.001
Change in dietary cholesterol from baseline (mg/day)				
6 mo	−71.8±312.2	−66.7±243.4	11.7±283.2	0.06
12 mo	−87.4±377.0	−78.8±239.8	1.76±325.3	0.09
24 mo	−94.2±373.5	−94.6±254.6	6.51±262.9‡	0.04
Change in dietary fiber from baseline (g/day)				
6 mo	−2.3±17.4	1.2±12.6	−7.4±22.0‖	0.002
12 mo	−4.3±19.0	0.22±12.9	−8.3±21.6‖	0.004
24 mo	−4.7±18.8	0.29±13.8	−10.0±21.7‖	0.002
Level of physical activity (MET/wk)				
Baseline	15.8±22.6	11.3±15.7	14.0±29.4	0.37
6 mo	17.5±23.1	14.2±19.3	18.7±32.0	0.41
12 mo	20.0±23.6	16.1±18.0	18.8±32.5	0.55
24 mo	21.4±26.7	15.6±18.9	16.3±18.9	0.18
Detectable urinary ketone bodies (% of participants)				
Baseline	2.9	1.8	4.6	0.10
24 mo	4.8	2.8	8.3	0.04

* Plus–minus values are means ±SD.

† P values for differences among the three diet groups were calculated by analysis of variance, except for urinary ketone values, for which the chi-square test was used. When the difference among the groups was significant (P<0.05), all pairwise comparisons between groups were tested for significance with the use of Tukey's Studentized range test. The Mediterranean-diet group consumed the largest amounts of dietary fiber and had the highest ratio of monounsaturated to saturated fat (P<0.05 for all comparisons among treatment groups). The low-carbohydrate group consumed the smallest amount of carbohydrates and the largest amounts of fat, protein, and cholesterol; the percentage of participants with detectable urinary ketones was also highest in this group (P<0.05 for all comparisons among treatment groups). The amount of decrease in intake of calories was similar among the diet groups.

‡ The value for the low-carbohydrate group is significantly different from the value for the low-fat group or the Mediterranean-diet group (P<0.05).

§ The value for the low-carbohydrate group is significantly different from the value for the low-fat group (P<0.05).

¶ The value for the Mediterranean-diet group is significantly different from the value for the low-fat group or the low-carbohydrate group (P<0.05).

‖ The value for the low-carbohydrate group is significantly different from the value for the Mediterranean-diet group (P<0.05).

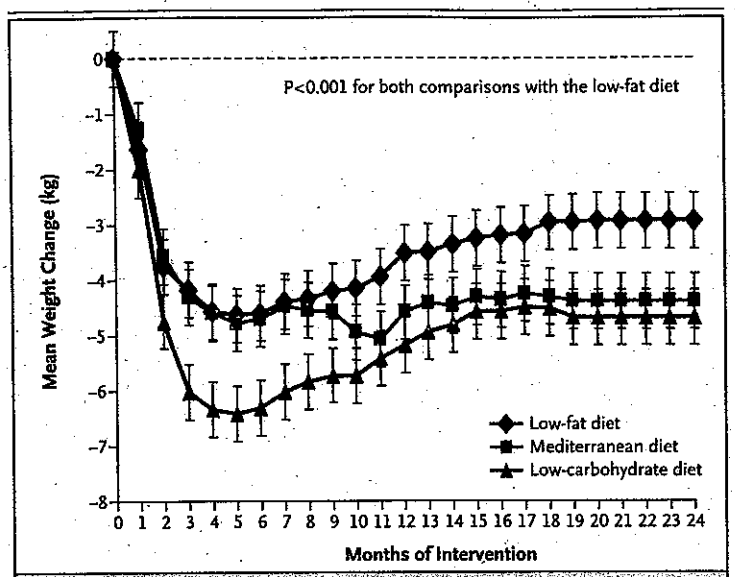

Figure 2. Weight Changes during 2 Years According to Diet Group.
Vertical bars indicate standard errors. To statistically evaluate the changes in weight measurements over time, generalized estimating equations were used, with the low-fat group as the reference group. The explanatory variables were age, sex, time point, and diet group.

cant ($P<0.05$) only in the low-carbohydrate group ($P=0.45$ for the comparison among groups).

LIVER-FUNCTION TESTS

Changes in bilirubin, alkaline phosphatase, and alanine aminotransferase levels were similar among the diet groups. Alanine aminotransferase levels were significantly reduced from baseline to 24 months in the Mediterranean-diet and the low-carbohydrate groups (reductions of 3.4±11.0 and 2.6±8.6 units per liter, respectively; $P<0.05$ for the comparison with baseline in both groups).

DISCUSSION

In this 2-year dietary-intervention study, we found that the Mediterranean and low-carbohydrate diets are effective alternatives to the low-fat diet for weight loss and appear to be just as safe as the low-fat diet. In addition to producing weight loss in this moderately obese group of participants, the low-carbohydrate and Mediterranean diets had some beneficial metabolic effects, a result suggesting that these dietary strategies might be considered in clinical practice and that diets might be individualized according to personal preferences and metabolic needs. The similar caloric deficit achieved in all diet groups suggests that a low-carbohydrate, non–restricted-calorie diet may be optimal for those who will not follow a restricted-calorie dietary regimen. The increasing improvement in levels of some biomarkers over time up to the 24-month point, despite the achievement of maximum weight loss by 6 months, suggests that a diet with a healthful composition has benefits beyond weight reduction.

The present study has several limitations. We enrolled few women; however, we observed a significant interaction between the effects of diet group and sex on weight loss (women tended to lose more weight on the Mediterranean diet), and this difference between men and women was also reflected in the changes in leptin levels. This possible sex-specific difference should be explored in further studies. The data from the few participants with diabetes are of interest, but we recognize that measurement of HOMA-IR is not an optimal method to assess insulin resistance among persons with diabetes. We relied on self-reported dietary intake, but we validated the dietary assessment in two different dietary-assessment tools and used electronic questionnaires to minimize the amount of missing data. Finally, one might argue that the unique nature of the workplace in this study, which permitted a closely monitored dietary intervention for a period of 2 years, makes it difficult to generalize the results to other free-living populations. However, we believe that similar strategies to maintain adherence could be applied elsewhere.

The strengths of the study include the one-phase design, in which all participants started simultaneously; the relatively long duration of the study; the large study-group size; and the high rate of adherence. The monthly measurements of weight permitted a better understanding of the weight-loss trajectory than was the case in previous studies.

We observed two phases of weight change: initial weight loss and weight maintenance. The maximum weight reduction was achieved during the first 6 months; this period was followed by the maintenance phase of partial rebound and a plateau. Among all diet groups, weight loss was greater for those who completed the 24-month study than for those who did not. Even moderate weight loss has health benefits, and our findings suggest benefits of behavioral approaches that yield weight losses similar to those obtained with pharmacotherapy.[29]

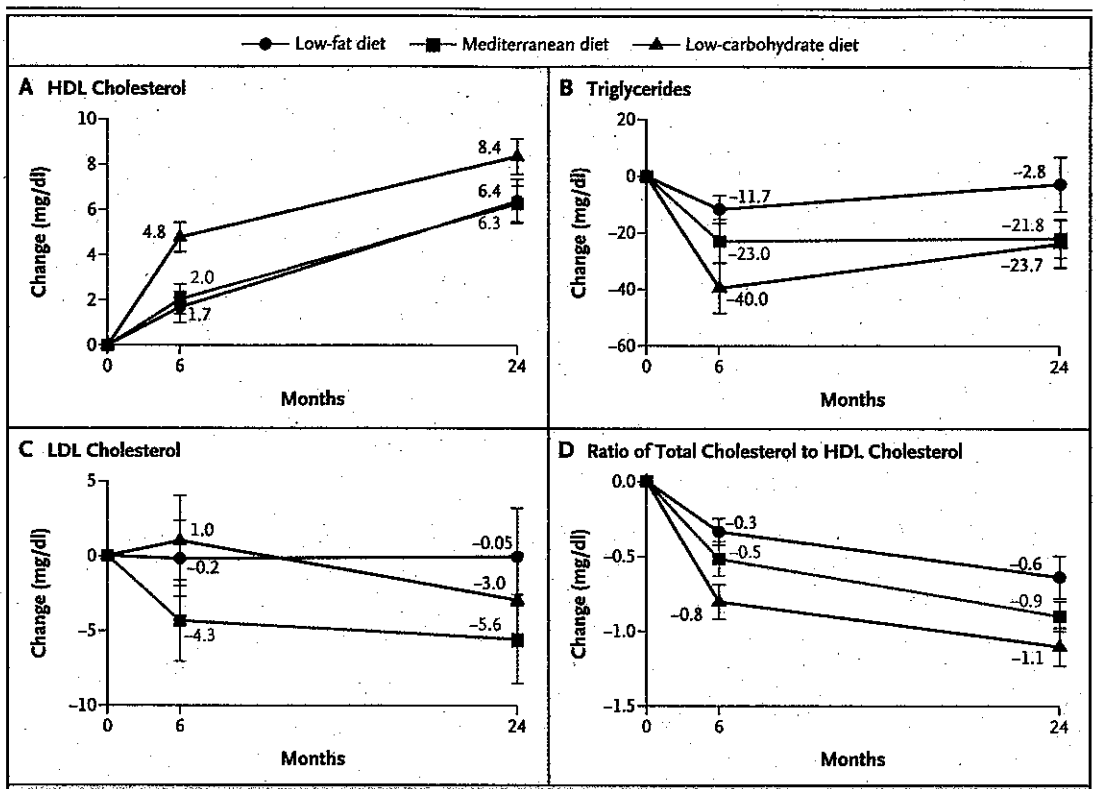

Figure 3. Changes in Cholesterol and Triglyceride Biomarkers According to Diet Group during the Maximum Weight-Loss Phase (1 to 6 Months) and the Weight-Loss Maintenance Phase (7 to 24 Months) of the 2-Year Intervention. Panel A shows the results for serum high-density lipoprotein (HDL) cholesterol, Panel B for serum triglycerides, Panel C for serum low-density lipoprotein (LDL) cholesterol, and Panel D for the ratio of total cholesterol to HDL cholesterol. Vertical bars indicate standard deviations. To statistically evaluate the changes in weight measurements over time, generalized estimating equations were used, with the low-fat group as the reference group. The explanatory variables were age, sex, time point, and diet group. Results are presented for the 82% of the study population (263 participants) with blood-sample data at all time points (90 in the low-fat group, 92 in the Mediterranean-diet group, and 81 in the low-carbohydrate group). The P values for the comparison between the low-fat group and the Mediterranean-diet group are 0.94 for HDL cholesterol, 0.21 for triglycerides, 0.41 for LDL cholesterol, and 0.23 for the ratio of total cholesterol to HDL cholesterol. The P values for the comparison between the low-fat group and the low-carbohydrate group are 0.01 for HDL cholesterol, 0.03 for triglycerides, 0.94 for LDL cholesterol, and 0.01 for the ratio of total cholesterol to HDL cholesterol. To convert values for cholesterol to millimoles per liter, multiply by 0.02586. To convert values for triglycerides to millimoles per liter, multiply by 0.01129.

We distinguished between changes in levels of biomarkers (leptin, adiponectin, and high-sensitivity C-reactive protein) that are apparently related to loss of adipose tissue and changes in biomarkers (triglycerides, HDL cholesterol, glucose, and insulin) that apparently reflect, in part, the effects of specific diet composition. The changes we observed in levels of adiponectin and leptin,[30] which were consistent in all groups, reflect loss of weight. Consumption of monounsaturated fats is thought to improve insulin sensitivity,[14,31,32] an effect that may explain the favorable effect of the Mediterranean diet on glucose and insulin levels. The results imply that dietary composition modifies metabolic biomarkers in addition to leading to weight loss. Our results suggest that health care professionals might consider more than one dietary approach, according to individual preferences and metabolic needs, as long as the effort is sustained.

This trial also suggests a model that might be applied more broadly in the workplace. As Okie recently suggested,[33] using the employer as a health coach could be a cost-effective way to improve health. The model of intervention with the use of dietary group sessions, spousal sup-

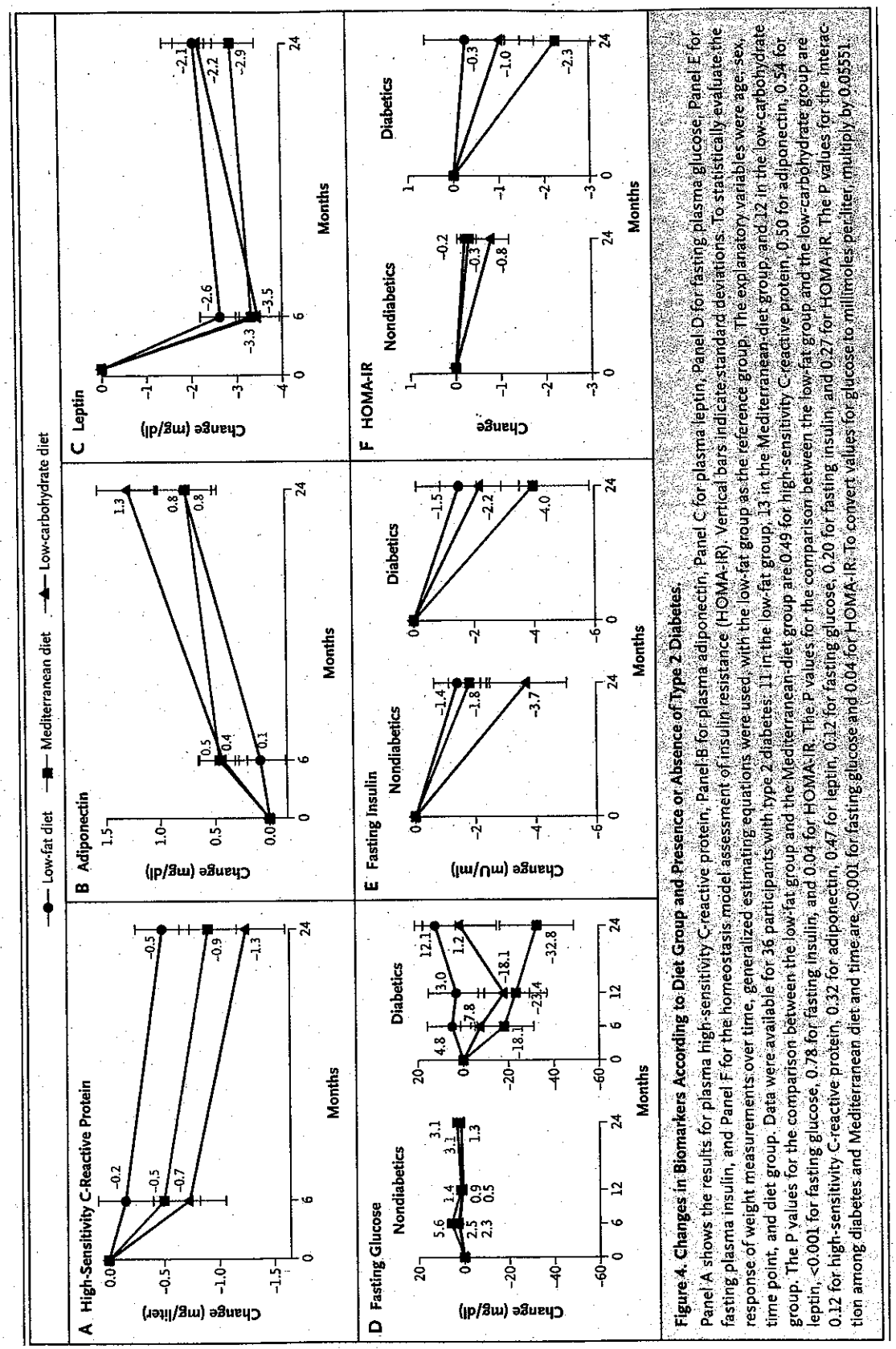

Figure 4. Changes in Biomarkers According to Diet Group and Presence or Absence of Type 2 Diabetes.

Panel A shows the results for plasma high-sensitivity C-reactive protein, Panel B for plasma adiponectin, Panel C for plasma leptin, Panel D for fasting plasma glucose, Panel E for fasting plasma insulin, and Panel F for the homeostasis model assessment of insulin resistance (HOMA-IR). Vertical bars indicate standard deviations. To statistically evaluate the response of weight measurements over time, generalized estimating equations were used, with the low-fat group as the reference group. The explanatory variables were age, sex, time point, and diet group. Data were available for 36 participants with type 2 diabetes: 11 in the low-fat group, 13 in the Mediterranean-diet group, and 12 in the low-carbohydrate group. The P values for the comparison between the low-fat group and the Mediterranean-diet group are 0.49 for high-sensitivity C-reactive protein, 0.50 for adiponectin, 0.54 for leptin, <0.001 for fasting glucose, 0.78 for fasting insulin, and 0.04 for HOMA-IR. The P values for the comparison between the low-fat group and the low-carbohydrate group are 0.12 for high-sensitivity C-reactive protein, 0.32 for adiponectin, 0.47 for leptin, 0.12 for fasting glucose, 0.20 for fasting insulin, and 0.27 for HOMA-IR. The P values for the interaction among diabetes and Mediterranean diet and time are <0.001 for fasting glucose and 0.04 for HOMA-IR. To convert values for glucose to millimoles per liter, multiply by 0.05551.

port, food labels, and monthly weighing in the workplace within the framework of a health promotion campaign might yield weight reduction and long-term health benefits.

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Exhibit E

The American Journal of Clinical Nutrition

Original Research Communications

Dietary energy density in the treatment of obesity: a year-long trial comparing 2 weight-loss diets[1–3]

Julia A Ello-Martin, Liane S Roe, Jenny H Ledikwe, Amanda M Beach, and Barbara J Rolls

ABSTRACT

Background: Consuming foods low in energy density (kcal/g) decreases energy intake over several days, but the effectiveness of this strategy for weight loss has not been tested.

Objective: The effects on weight loss of 2 strategies for reducing the energy density of the diet were compared over 1 y.

Design: Obese women ($n = 97$) were randomly assigned to groups counseled either to reduce their fat intake (RF group) or to reduce their fat intake and increase their intake of water-rich foods, particularly fruit and vegetables (RF+FV group). No goals for energy or fat intake were assigned; the subjects were instructed to eat ad libitum amounts of food while following the principles of their diet.

Results: After 1 y, study completers ($n = 71$) in both groups had significant decreases in body weight ($P < 0.0001$). Subjects in the RF+FV group, however, had a significantly different pattern of weight loss ($P = 0.002$) than did subjects in the RF group. After 1 y, the RF+FV group lost 7.9 ± 0.9 kg and the RF group lost 6.4 ± 0.9 kg. Analysis of all randomly assigned subjects also showed a different pattern of weight loss between groups ($P = 0.021$). Diet records indicated that both groups had similar reductions in fat intake. The RF+FV group, however, had a lower dietary energy density than did the RF group ($P = 0.019$) as the result of consuming a greater weight of food ($P = 0.025$), especially fruit and vegetables ($P = 0.037$). The RF+FV group also reported less hunger ($P = 0.003$).

Conclusion: Reducing dietary energy density, particularly by combining increased fruit and vegetable intakes with decreased fat intake, is an effective strategy for managing body weight while controlling hunger. *Am J Clin Nutr* 2007;85:1465–77.

KEY WORDS Energy density, fruit and vegetables, water-rich foods, fat intake, obesity, weight management

INTRODUCTION

With the prevalence of obesity doubling since 1980 (1), there is an urgent need for effective, nutritionally balanced weight-loss strategies. Although many diets produce short-term weight loss, long-term dietary adherence and maintenance of weight loss are difficult to achieve (2). Approaches to reduce energy intake typically focus on limiting portions or food choices; however, such restrictive approaches may lead to hunger and dissatisfaction. In clinical trials, hunger has been associated with a lack of weight loss or with weight regain (3–5). Conversely, a dietary strategy that helps individuals control hunger by eating satisfying amounts of food could improve adherence and increase weight loss. One such approach is to encourage individuals to decrease the energy density of the diet by consuming low-energy-density foods.

The energy density (kcal/g) of the diet can be decreased by reducing intake of fat, which has a higher energy density (9 kcal/g) than does carbohydrate or protein (4 kcal/g). Incorporating water-rich foods such as fruit and vegetables into the diet can also reduce dietary energy density because water adds weight and volume to food without adding energy. For the same number of calories, individuals following a low-energy-density diet can eat a greater weight of food, and thus may experience less hunger than do individuals following a diet that restricts portions.

Multiple laboratory-based, controlled studies have been conducted in which participants were served test foods or diets that were varied in either fat content, the amount of fruit and vegetables, or both (6–13). The results of these studies indicated that these strategies to reduce dietary energy density were effective for decreasing energy intake for periods of up to 11 wk. Several clinical trials found that restricting fat consumption and combining fat reduction with increased fruit and vegetable intake both lead to weight loss (14–17). Although these trials did not assess the energy density of the diet (18), it is possible that the weight loss was related to a decrease in dietary energy density. The specific influence of dietary energy density on body weight, however, has not been extensively investigated. One trial that did assess both energy density and body weight found that instructing subjects to incorporate 2 servings of low-energy-density soup daily into a reduced-calorie diet led to greater weight loss than did incorporation of the same amount of energy as high-energy-density snack foods (19). These studies suggest that reducing the energy density of the diet is likely to be an effective strategy for weight management and should be explored further.

The present trial investigated the effects on weight loss of advice for reducing the energy density of the diet. Two different strategies were tested: one advised a reduction in fat intake and the other promoted an increased consumption of water-rich foods along with a reduction in fat intake. It was hypothesized that

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advising individuals to follow a reduced-fat diet would lead to a decrease in dietary energy density that would be associated with weight loss. In addition, it was hypothesized that promoting the incorporation of water-rich foods into a reduced-fat diet would lead to a further reduction in dietary energy density and energy intake, greater weight loss, and less hunger than would advising a reduced-fat diet alone.

SUBJECTS AND METHODS

Subjects

Potential subjects were recruited through flyers and newspaper advertisements. Eligibility was determined through a telephone interview, a physical screening, and questionnaires assessing symptoms of depression (20), symptoms of eating disorders (21–23), and the ability to safely engage in physical activity (24). Subjects were considered for inclusion in the study if they were women aged 20 to 60 y with a body mass index (BMI; in kg/m^2) of 30 to 40; thus, all subjects were classified as obese (25). Respondents were excluded if they had blood pressure >140/90 mm Hg, serum triacylglycerols >400 mg/dL, or total cholesterol higher than the 90th percentile for their age (26); had a serious medical condition that precluded participation or any condition limiting physical activity; were pregnant or lactating; were taking a selective serotonin re-uptake inhibitor; had symptoms of depression or disordered eating; or were participating in a weight-loss program.

The sample size required in the trial was estimated by using data from obese women who participated in a weight-loss trial utilizing a dietary intervention with a similar frequency of contact (19). It was estimated that a sample of 35 subjects per intervention group would allow the detection of a 2.4-kg (5.3-lb) difference in weight loss between the groups by use of a repeated-measures analysis with a significance level of 0.05 and 80% power. On the basis of the previous trial, a subject attrition rate of 30% was predicted; thus, the aim was to enroll 50 subjects per intervention group.

One hundred ten women met the initial inclusion criteria and participated in a two-week run-in period (27, 28). During this period, 97 women met the secondary inclusion criteria (completing paperwork and attending sessions). These subjects were classified by age and severity of obesity (BMI) and were randomly assigned to 1 of 2 intervention groups through the use of a stratified permuted block design (**Figure 1**). Because this was to be the first intervention of this type to assess energy density, only women were tested to simplify the design and to enhance statistical power. Subjects provided written informed consent and were financially compensated for their participation. The protocol was approved by the Office for Research Protections of The Pennsylvania State University, and the clinical trial was conducted at the General Clinical Research Center at the University Park campus. This study was registered in the public trial registry maintained by the US National Library of Medicine (http://www.clinicaltrials.gov) as "Factors Affecting Caloric Regulation in Human Feeding" (NCT00108784).

Study design

The year-long intervention was divided into 2 phases. During the first 6 mo (phase 1), participants met individually with a dietitian once per week, attending a maximum of 26 counseling sessions lasting ≈30 min. During the second 6 mo (phase 2), participants attended one small group session and one individual session with a dietitian each month. They attended a maximum of six 60-min small group sessions and six 15- to 30-min individual sessions during this phase.

Subjects in both intervention groups received the same amount of instruction on fat reduction, behavior change, physical activity, and the principles of their diets. To avoid counseling bias, individual dietitians spent a similar proportion of their time with subjects in each intervention group. Because the intervention required specific dietary changes, neither the participants nor the dietitians were blinded to intervention assignment. Although the participants were aware that there were 2 intervention groups, they were unaware of the dietary advice that was being provided to the other group.

Interventions

The goal for the subjects in both intervention groups was to reduce the energy density of their diets; however, the groups were taught different dietary strategies to achieve this goal. The reduced-fat (RF) group was advised to reduce fat intake, whereas the reduced-fat plus increased fruit and vegetable (RF+FV) group was advised to reduce fat intake and increase intake of water-rich foods, especially fruit and vegetables. Within each of the strategies, the subjects were taught to make food choices (reduced-fat foods or reduced-fat and water-rich foods) that were reduced in energy density and appropriate in portion size. Neither group was given daily limits for energy or fat intake; the subjects were instructed to eat ad libitum amounts of food while following the principles of their assigned diet.

Phase 1 (individual lessons)

During phase 1, the subjects received written materials and individual instruction from the dietitians. To achieve a reduced-fat diet, the subjects were instructed in cooking and recipe modifications and grocery shopping and dining-out strategies and were provided with meal and snack ideas. Subjects in the RF group were taught recommended serving sizes for common foods (29) and were advised to choose appropriate portion sizes. To match the 2 groups for the amount of instruction provided, the RF group was also counseled on nutrition topics important for women's health (eg, iron intake).

Subjects in the RF+FV group were instructed in the same methods of dietary change as were the subjects in the RF group, but strategies to increase water-rich foods (eg, fruit, vegetables, and soups) were included in addition to strategies to reduce fat. These subjects were also taught about recommended serving sizes (29), but were encouraged to eat larger, satisfying portions of low-energy-density foods (fruit, vegetables, and soups) and recommended serving sizes of medium- and high-energy-density foods (30).

Subjects in both groups received the same behavior therapy recommendations based on social cognitive theory (31, 32). Behavioral therapy emphasized increasing self-efficacy for lifestyle changes. Some key topics incorporated into the intervention were self-monitoring, goal setting, social support networks, coping with emotional eating, managing stress and the environment, overcoming obstacles, problem solving, and handling setbacks.

Physical activity information for both groups focused on walking and using a pedometer to set goals and track progress. Subjects' baseline step counts were determined over 7 days during

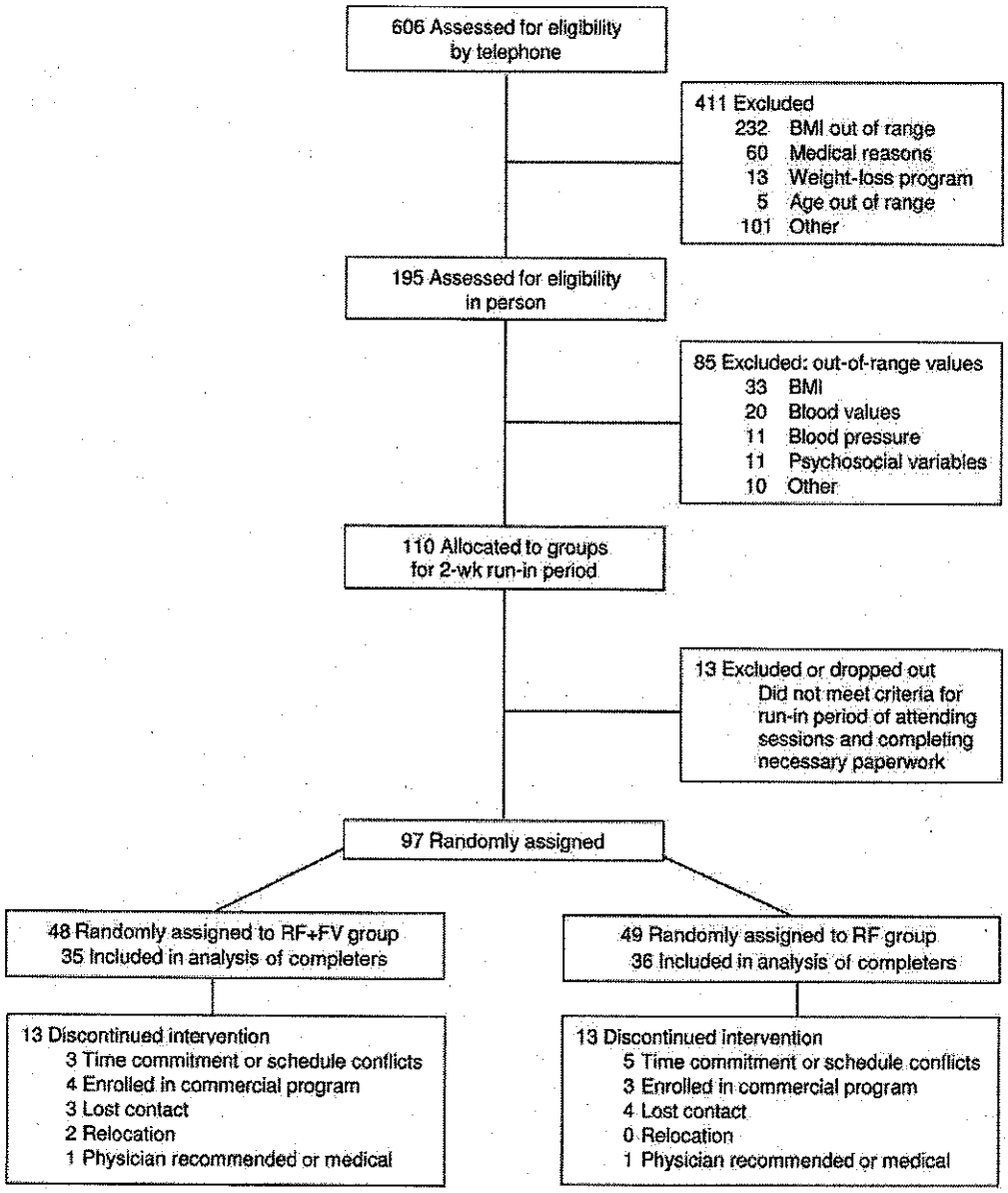

FIGURE 1. Flow diagram of subject enrollment, random assignment, and completion of the study protocol.

the first week of the study. Subjects set goals to gradually increase their step counts by 20% of their baseline measure or 2000 steps per day (33, 34). The long-term goal for subjects was to reach 10 000 steps per day.

Phase 2 (group and individual lessons)

During phase 2, the subjects met in small groups within their intervention group (RF or RF+FV) to review the material presented during phase 1. The group lessons, which were led by the dietitians, comprised 6 topics for practical review: holiday eating, cooking and recipe modification, appropriate portion sizes, label reading, dining out, and grocery shopping. Although the primary focus of the group sessions was on the diet, physical activity was encouraged and all subjects received a handout at the conclusion of each session with practical information related to activity. During monthly individual sessions, the dietitians met with the subjects to review their diet records and discuss any questions or concerns.

Assessment of study outcomes

Changes in body weight and body composition

At each session, the subjects were weighed (within 0.1 kg) without shoes and while wearing light clothing on a calibrated scale. Standing height (within 0.5 cm) was measured at baseline and was confirmed at month 6. Body-composition measures were taken at baseline and months 3, 6, and 12. Percentage of body fat (within 0.1%) was measured by using bioelectrical impedance (Biodynamics model 310; Biodynamics Corporation, Seattle, WA) after the subjects had fasted for 12 h. The day before the impedance measurement, the subjects were instructed to abstain from alcohol and to drink plenty of fluids to be well

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hydrated (35). Waist circumference was measured (within 0.5 cm) by using the protocol of the third National Health and Nutrition Examination Survey (36, 37).

Diet composition

Before starting the study, the subjects attended a training session that taught them how to properly complete 3-d diet records; the session included special instructions on recording foods and beverages so that energy density could be correctly calculated. The subjects completed detailed diet records for 3 consecutive days (2 weekdays and 1 weekend day) every 2 wk during phase 1 and every 4 wk during phase 2. During the counseling sessions, the dietitians reviewed the diet records with the subjects to promote completeness and accuracy.

The diet records were analyzed by the Diet Assessment Center at The Pennsylvania State University, which has experience in calculating energy density from food records by using the Nutrition Data System for Research (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN). In the analyses of total energy intake, all foods and caloric beverages were included. Fruit and vegetable intakes were analyzed both with and without those that were fried and dried to examine the effect of water-rich fruit and vegetables on weight loss.

Food energy density was calculated as the ratio between food energy (kcal) and food weight (g), excluding caloric beverages (such as milk and juices) as well as noncaloric beverages. The inclusion of beverages may disproportionately influence the calculation of energy density because beverages tend to be much lower in energy density than are most foods (38). Epidemiologic data indicate that including beverages may diminish associations between energy density and outcome variables because of increased within-person variance (38).

Hunger and satiety

On the same days that the subjects completed their diet records, they also rated their daily hunger and fullness 1.5 h after their evening meal (39–41) by using 100-mm visual analogue scales. For example, hunger was assessed with the question "How hungry did you feel today?"; the scale was anchored on the left by "not at all hungry" and on the right by "extremely hungry." Hunger and fullness were also rated immediately before and after each meal (breakfast, lunch, and dinner) on the first day that subjects completed diet records. Meal ratings were collected to assess the reliability of the single daily ratings.

Physical activity

Walking, the physical activity emphasized in the intervention, was measured by daily step counts. Subjects were provided with calibrated pedometers and were instructed in their use. They were required to record a minimum of 3 d of step counts every 2 wk in phase 1 and once per month in phase 2. The subjects recorded their step counts on the same 3 days that they recorded their food intake and rated their hunger and satiety.

Diet satisfaction

Subjects completed a Diet Satisfaction Questionnaire at baseline and months 3, 6, and 12 (42). The details of the development and validation of this instrument will be presented in a separate publication. Briefly, this questionnaire evaluates a variety of issues identified from the literature that may affect satisfaction with the diet. It assesses the following 7 factors, which were determined by principal components analyses: family dynamics, cost, preparation, convenience, healthy lifestyle, negative aspects, and preoccupation with food. The questionnaire provides a score for each of the factors as well as a global score for overall diet satisfaction. Items are coded so that a higher score indicates greater satisfaction or perceived benefit.

Psychosocial factors

The subjects completed several questionnaires at baseline, 6 mo, and 12 mo, including the Eating Inventory (43) to measure dietary restraint (the tendency to consciously restrict food intake to control body weight), disinhibition (the loss of control over eating in response to emotional or social cues), and hunger (the tendency to subjective feelings of hunger). At the same time points, the subjects also completed the Beck Depression Inventory II (20), which measures symptoms of depression in obese individuals, and the Eating Habits Checklist (23), which assesses the extent to which obese individuals experience binge eating problems.

Laboratory analyses and blood pressure

Blood samples were taken and analyzed for lipids and insulin at baseline and at 3, 6, and 12 mo and were analyzed for carotenoids at baseline, 6 mo, and 12 mo. The subjects were required to fast for 12 h and to abstain from alcohol for 48 h before providing a blood sample. Insulin and lipids were analyzed by Clinical Laboratory Services, Penn State Milton S Hershey Medical Center. Serum concentrations of total cholesterol and triacylglycerol were measured by enzymatic procedures (44). HDL cholesterol was measured after the precipitation of apolipoprotein B–containing lipoproteins in whole plasma by heparin-manganese chloride (45); LDL-cholesterol concentrations were calculated with use of the Friedewald equation (46). Carotenoids were analyzed by the University of California, San Diego, Cancer Center Nutrition Shared Resource. Plasma carotenoids were separated and quantified with an HPLC method (47). Blood pressure was measured twice per counseling session according to a standard protocol (48).

Statistical methods

All outcomes were analyzed by using a mixed model that included intervention group as a fixed effect. For the outcomes that were measured at many time points, including body weight, a random coefficients analysis was used to model the longitudinal response over time (49). Time was treated as a continuous covariate in the model, and lower-order polynomial factors of time were fitted if they were significantly related to the outcome. This model accounts for the correlation of the repeated measures within subjects by allowing the longitudinal response to vary randomly for each subject. Thus, the model can estimate changes in outcomes on the basis of all observations, including data for subjects who withdraw from the trial (50). By use of this method, the outcome of body weight was analyzed both for study completers and for all randomly assigned subjects. For outcomes such as dietary intakes and blood lipids, which were measured at few time points, the within-subject correlation was accounted for by using a covariance pattern in the mixed model. For all outcomes, the baseline value was included as a covariate; additionally, serum total cholesterol and BMI were included as covariates for the analysis of blood carotenoids (51).

TABLE 1
Baseline characteristics in the 2 intervention groups of all randomly assigned subjects and for subjects who completed the trial[1]

	All randomly assigned subjects		Study completers	
Characteristic	RF group (n = 49)	RF + FV group (n = 48)	RF group (n = 36)	RF + FV group (n = 35)
Age (y)	44.5 ± 1.3[2]	45.3 ± 1.4	45.1 ± 1.5	46.7 ± 1.5
Ethnicity [n (%)]				
White	44 (90)	47 (97)	33 (92)	34 (97)
Other	5 (10)	1 (3)	3 (8)	1 (3)
Educational level [n (%)]				
High school diploma or less	14 (28)	12 (25)	10 (28)	9 (26)
Some college to associate degree	18 (37)	15 (31)	11 (30)	10 (28)
Bachelor's to graduate degree	17 (35)	21 (44)	15 (42)	16 (46)
Hunger score[3]	6.0 ± 0.4	6.1 ± 0.5	6.0 ± 0.5	6.1 ± 0.6
Disinhibition score[3]	9.6 ± 0.5	10.0 ± 0.5	9.7 ± 0.6	10.2 ± 0.6
Dietary restraint score[3]	9.4 ± 0.6	8.9 ± 0.5	9.5 ± 0.7	9.0 ± 0.6
Depression score[4]	7.5 ± 0.9	6.8 ± 0.8	7.0 ± 1.0	6.9 ± 1.0
Binge-eating score[5]	12.6 ± 0.9	13.3 ± 0.9	11.8 ± 1.0	13.1 ± 0.9

[1] RF, reduced-fat intervention; RF + FV, reduced-fat plus increased fruit and vegetable intervention. Baseline values did not differ significantly between groups (unpaired t test), either for all randomly assigned subjects or for subjects who completed the trial.
[2] $\bar{x}$ ± SEM (all such values).
[3] Eating Inventory (40).
[4] Beck Depression Inventory (18).
[5] Eating Habits Checklist (21).

Multivariate ANOVA was used to test macronutrient intakes as a percentage of total energy intake. Step-wise regression analyses were performed to predict weight loss on the basis of dietary measures (energy intake, energy density of food, total fat intake, combined fruit and vegetable intake, and total weight of food), physical activity (step counts), and scores from the Eating Inventory (dietary restraint, disinhibition, and hunger) at months 1, 2, 3, 6, 9, and 12. All analyses were performed by using SAS software (version 9; SAS Institute Inc, Cary, NC). Results were considered significant at $P < 0.05$; values are presented as means ± SEMs.

RESULTS

Subjects

There were no significant differences at baseline in characteristics of the subjects assigned to the 2 intervention groups (**Table 1**). Subjects ranged in age from 22 to 60 y, and 76% were employed full-time. At 12 mo, 71 subjects (73%) remained in the trial; these subjects are referred to as the study completers (Figure 1). The reasons given most frequently for withdrawal were inability to attend the counseling sessions because of schedule conflicts and relocation out of the area. There were no significant differences in baseline characteristics, including body weight, between the participants who completed the trial and those who withdrew.

Changes in body weight and body composition

After 1 y, study completers in both intervention groups (n = 71) had lost a significant amount of weight compared with their baseline values ($P < 0.0001$; **Table 2**). Over the course of the trial, however, study completers who were advised to reduce their dietary fat intake and increase their intake of water-rich foods (RF+FV group) had a significantly different pattern of weight loss than did subjects who were advised to reduce their fat intake alone (RF group) ($P = 0.002$ for group × time interaction; **Figure 2**). During the first 6 mo of the trial (phase 1), completers in the RF+FV group lost more body weight (8.9 ± 0.8 kg, or 19.6 ± 1.8 lb) than did those in the RF group (6.7 ± 0.7 kg, or 14.7 ± 1.5 lb; $P = 0.034$), a difference of 33%. During the second 6 mo of the trial (phase 2), the pattern of weight change did not differ significantly between the groups ($P = 0.056$). In phase 2, when subject contact was less frequent, weight was well maintained; subjects in both groups regained an average of 0.7 ± 0.4 kg (1.5 ± 0.9 lb). At the end of the trial, mean weight loss was 7.9 ± 0.9 kg (17.4 ± 1.9 lb) in the RF+FV group and 6.4 ± 0.9 kg (14.1 ± 1.9 lb) in the RF group.

The analysis of all randomly assigned subjects (n = 97) yielded conclusions similar to the analysis of study completers: over the course of the year, subjects in the RF+FV group lost significantly more body weight than did subjects in the RF group ($P = 0.021$). When all available data were included, the estimate of weight loss at the end of the trial was smaller in both groups (6.4 ± 0.8 kg in the RF+FV group and 4.9 ± 0.8 kg in the RF group). As in the analysis of completers, during phase 1 of the trial, there was a greater decline in body weight in the RF+FV group than in the RF group ($P = 0.003$), and during phase 2, the pattern of weight change did not differ significantly between the groups ($P = 0.074$).

Over the course of the study, study completers in both groups showed significant decreases in BMI, percentage body fat, and waist circumference from baseline values ($P \leq 0.0001$; Table 2). Subjects in the RF+FV group had a significantly different pattern in the change in BMI than did subjects in the RF group ($P < 0.0001$ for group × time interaction). During phase 1, completers in the RF+FV group had a greater decrease in BMI than did those in the RF group (−3.3 ± 0.3 versus −2.5 ± 0.3; $P = 0.0001$), whereas during phase 2, changes in BMI did not differ significantly between the groups (−2.9 ± 0.4 in the RF+FV group and

TABLE 2
Body-composition measures in study completers over the course of 1 y[1]

Outcome at selected time points	RF group ($n = 36$) Value	RF group ($n = 36$) Change from baseline value	RF + FV group ($n = 35$) Value	RF + FV group ($n = 35$) Change from baseline value	Difference between groups Effect	Difference between groups P
Body weight (kg)					Group × time[2]	0.002
Baseline	90.2 ± 1.4[3,4]	—	90.8 ± 1.8[4]	—		
3 mo	85.3 ± 1.4	−4.9 ± 0.5	84.3 ± 1.7	−6.5 ± 0.5		
6 mo	83.5 ± 1.5	−6.7 ± 0.7	81.9 ± 1.7	−8.9 ± 0.8		
12 mo	83.8 ± 1.7	−6.4 ± 0.9	82.9 ± 2.0	−7.9 ± 0.9		
BMI (kg/m^2)					Group × time[2]	<0.0001
Baseline	33.3 ± 0.4[3,4]	—	33.4 ± 0.5[4]	—		
3 mo	31.4 ± 0.4	−1.8 ± 0.2	31.0 ± 0.5	−2.4 ± 0.2		
6 mo	30.8 ± 0.4	−2.5 ± 0.3	30.2 ± 0.5	−3.3 ± 0.3		
12 mo	30.9 ± 0.5	−2.4 ± 0.4	30.5 ± 0.6	−2.9 ± 0.4		
Body fat (%)					Group[5]	0.71
					Time	<0.0001
Baseline	39.2 ± 0.4[3,4]	—	38.8 ± 0.5[4]	—		
3 mo	37.6 ± 0.4	−1.6 ± 0.3	37.0 ± 0.5	−1.8 ± 0.3		
6 mo	36.5 ± 0.5	−2.7 ± 0.4	35.6 ± 0.7	−3.2 ± 0.5		
12 mo	36.6 ± 0.6	−2.6 ± 0.5	36.2 ± 0.7	−2.6 ± 0.5		
Body fat (kg)					Group[5]	0.44
					Time	<0.0001
Baseline	35.4 ± 0.7[3,4]	—	35.4 ± 1.0[4]	—		
3 mo	33.4 ± 0.7	−1.9 ± 0.2	32.9 ± 1.0	−2.6 ± 0.2		
6 mo	32.7 ± 0.7	−2.7 ± 0.3	31.9 ± 1.0	−3.5 ± 0.3		
12 mo	32.9 ± 0.8	−2.5 ± 0.4	32.4 ± 1.1	−3.1 ± 0.4		
Lean body mass (kg)					Group[5]	0.42
					Time	0.14
Baseline	54.6 ± 0.7[3,4]	—	55.0 ± 1.0[4]	—		
3 mo	53.1 ± 0.8	−1.4 ± 0.3	53.1 ± 0.8	−1.9 ± 0.6		
6 mo	52.9 ± 0.9	−1.6 ± 0.4	52.5 ± 0.8	−2.5 ± 0.6		
12 mo	52.9 ± 0.9	−1.6 ± 0.4	52.6 ± 0.9	−2.4 ± 0.7		
Waist circumference (cm)					Group[5]	0.27
					Time	0.0002
Baseline	106.5 ± 1.2[3,4]	—	106.0 ± 1.8[4]	—		
3 mo	102.8 ± 1.2	−3.7 ± 0.8	102.1 ± 1.5	−3.9 ± 1.1		
6 mo	104.1 ± 1.3	−2.4 ± 1.2	101.6 ± 1.7	−4.4 ± 1.3		
12 mo	99.7 ± 1.2	−6.8 ± 1.1	97.8 ± 1.9	−8.2 ± 1.3		

[1] All values are $\bar{x}$ ± SEM. RF, reduced-fat intervention; RF + FV, reduced-fat plus increased fruit and vegetable intervention.
[2] Random coefficients analysis was used to model the outcome across the 39 time points with control for baseline values. A significant group × time interaction indicates that the response over time differed between the groups. When all randomly assigned subjects ($n = 97$) were included in the analysis, the group × week interaction remained significant for body weight ($P = 0.021$) and BMI ($P = 0.024$).
[3] Baseline values did not differ significantly between groups (unpaired t test).
[4] Baseline values differed significantly from those at intervention time points according to a mixed model with repeated measures ($P \leq 0.0001$). This indicates a within-group effect of time when the group × time interaction was significant, and a main effect of time when there was no significant interaction.
[5] Differences in outcome between groups and over time were determined from a mixed linear model with repeated measures that included all available time points and that was controlled for baseline values. The group × time interaction was not significant.

−2.4 ± 0.4 in the RF group; $P = 0.514$). After 1 y of intervention, 49% of subjects in the RF+FV group were no longer classified as obese (BMI ≥ 30) compared with 28% of subjects in the RF group ($P = 0.071$). Measured body fat decreased by an average of 7% for subjects in both groups. Apart from differences in weight loss and BMI, there were no significant differences between the groups in body-composition measures.

Reported diet composition, hunger and satiety, and physical activity

Examination of diet records indicated that study completers in both groups made dietary changes that resulted in a significant decrease in the energy density of their diets ($P < 0.0001$; **Figure 3**). Participants in the RF+FV group, however, reduced the energy density of their diets during the intervention to a greater extent (1.23 ± 0.02 kcal/g) than did those in the RF group (1.46 ± 0.02 kcal/g; $P = 0.019$). Diet records showed that subjects in both groups significantly reduced their fat intake from 34.5 ± 0.6% of energy at baseline to 28.4 ± 0.4% during the intervention ($P \leq 0.0001$). Fat intake did not differ significantly between the groups ($P = 0.50$; **Table 3**); this was expected because both groups received the same advice for dietary fat reduction. Similarly, reported intakes of carbohydrates and protein did not differ significantly between the groups during the intervention; the groups consumed a mean of 54.4 ± 0.5% of energy from carbohydrate and 18.0 ± 0.2% of energy from protein. The groups differed, however, in reported fruit and vegetable intake; this was expected because the dietary advice concerning fruit and vegetables differed between the groups. Subjects in the RF+FV group reported a greater intake of fruit and vegetables ($P =$

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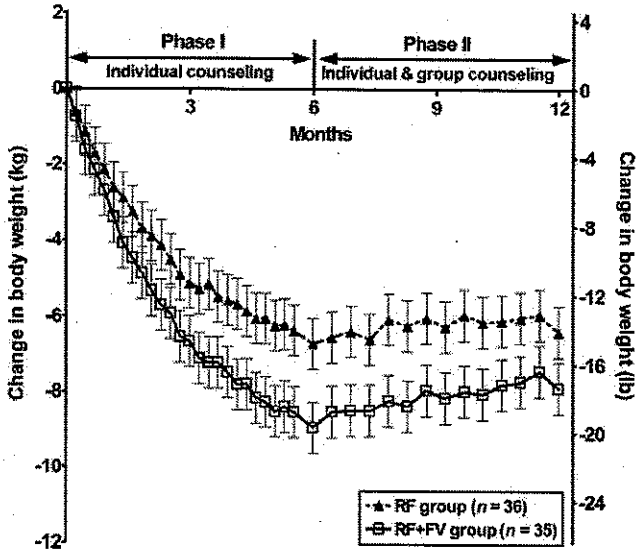

FIGURE 2. Mean (± SEM) change in body weight for study completers in the reduced-fat (RF) and reduced-fat plus increased fruit and vegetable (RF+FV) intervention groups over time. Random coefficients analysis was used to model the longitudinal response over time, with control for baseline values. The group × time interaction ($P = 0.002$) indicates that the response over time differed between the groups. The interaction remained significant ($P = 0.021$) when all randomly assigned subjects were included in the analysis. Baseline values did not differ significantly between the groups (unpaired t test).

0.037; Table 3) than did those in the RF group; this was true regardless of whether fried and dried fruit and vegetables were included in the analysis. Subjects in the RF+FV group also consumed significantly more dietary fiber (g/1000 kcal; $P = 0.001$).

Diet records during the intervention showed a reduction in energy intake from baseline values of ≈500 kcal/d ($P < 0.0001$; Table 3), which did not differ significantly between the groups. The difference in weight loss between the groups corresponded with an estimated difference in energy intake of ≈100 kcal/d, whereas the SE of the difference in energy intake between the groups was 113 kcal. Thus, given the variability in reported energy intake, we were not able to detect a difference in energy intake between the groups that would account for the difference in weight loss.

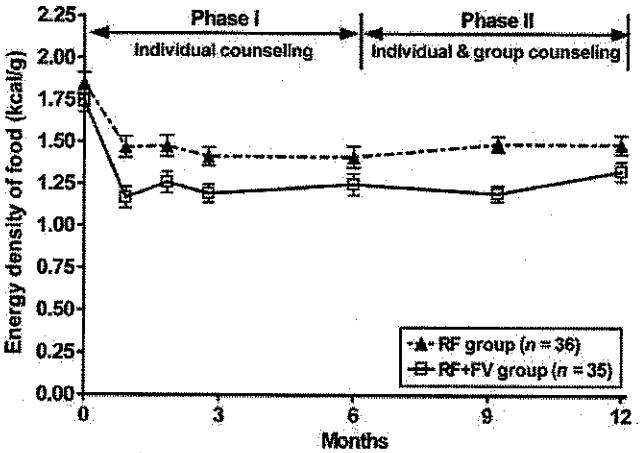

FIGURE 3. Mean (± SEM) food energy density (kcal/g) for study completers in the reduced-fat (RF) and reduced-fat plus increased fruit and vegetable (RF+FV) intervention groups over time. Differences in dietary energy density between the groups were determined from a mixed linear model with repeated measures, by using all available time points and with control for baseline values. The group × time interaction was significant ($P = 0.019$), which indicated that dietary energy density was significantly less in the RF+FV group than in the RF group during the intervention. Baseline values did not differ significantly between the groups (unpaired t test).

There was a significant difference between the groups in the weight of food consumed during the trial. Subjects in the RF+FV group reported consuming significantly more food than did subjects in the RF group ($P = 0.025$; Table 3). Specifically, the subjects who were encouraged to eat satisfying portions of low-energy-density, water-rich foods (RF+FV group) consumed a mean of 225 g (25%) more food daily.

Daily ratings of hunger, as measured by visual analogue scales at the end of the day, did not differ significantly from baseline for subjects in the RF group. Over the course of the study, however, hunger ratings reported by subjects in the RF+FV group were significantly lower than their ratings at baseline ($P = 0.030$). This resulted in significantly different ratings for hunger between the groups during the intervention (RF+FV group: 46.7 ± 2.2; RF group: 53.5 ± 2.2; $P = 0.003$). Ratings for fullness did not differ significantly between the groups. Analyses to assess the reliability of the daily measures showed that daily hunger ratings were significantly correlated with hunger ratings collected before and after meals; these results will be reported in a separate publication.

Physical activity, as measured by step counts, increased significantly from baseline for subjects in both groups ($P < 0.0001$; Table 3). Step counts did not differ significantly between the groups during the trial ($P = 0.50$); this was expected because the advice given for physical activity was the same for both groups. Subjects in both groups increased their daily step counts by an additional 2427 ± 100 steps/d to a total of 8735 ± 70 steps/d over the course of the trial. This exceeded the goal of an additional 2000 steps/d and increased the average daily step count by 38% from baseline, an increase that was maintained over the course of the year.

Questionnaires

Overall scores for satisfaction were significantly higher for the intervention diets (3.6 ± 0.1) than for the baseline diets (3.2 ± 0.1) for subjects in both groups (maximum score 5.0; $P < 0.0001$). There were no significant differences between the 2 dietary approaches in any of the ratings of diet satisfaction. Subjects in both groups rated their intervention diets as providing greater health benefits, having fewer negative aspects, working better within the family context, and leading to less preoccupation with food than did their baseline diets ($P < 0.0001$). Ratings of perceived cost effectiveness, preparation time, and convenience were lower for both diet strategies than for the diets consumed at baseline (all $P < 0.05$), but there were no significant differences between the groups.

Although there were significant changes from baseline on scores from the Eating Inventory, the Beck Depression Inventory II, and the Eating Habits Checklist, there were no significant differences between the groups. Analysis of the Eating Inventory showed that for all subjects there was a significant increase in dietary restraint scores at 6 and 12 mo (mean score 14.7 ± 0.3;

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TABLE 3
Dietary measures in study completers over the course of 1 y[1]

	RF group (n = 36)		RF + FV group (n = 35)		Difference between groups	
Outcome	Value	Change from baseline value	Value	Change from baseline value	Effect	P
Energy intake of food and beverages (kcal/d)					Group[2]	0.71
					Time	<0.0001
Baseline	1836 ± 68[3,4]	—	1937 ± 78[4]	—		
1 mo	1430 ± 60	−406 ± 84	1376 ± 47	−562 ± 76		
2 mo	1384 ± 62	−452 ± 87	1336 ± 64	−601 ± 85		
3 mo	1374 ± 62	−462 ± 79	1358 ± 60	−580 ± 81		
6 mo	1266 ± 62	−570 ± 79	1440 ± 60	−498 ± 81		
9 mo	1351 ± 71	−486 ± 89	1388 ± 76	−549 ± 98		
12 mo	1307 ± 62	−525 ± 80	1437 ± 60	−501 ± 81		
Food intake (g/d)					Group × time[2]	0.025
Baseline	975 ± 47[3]	—	1068 ± 49	—		
1 mo	936 ± 43[5]	−39 ± 44	1192 ± 68	124.6 ± 52		
2 mo	894 ± 42[5]	−81 ± 38	1101 ± 72	33.3 ± 62		
3 mo	936 ± 47	−39 ± 48	1117 ± 69	49.0 ± 54		
6 mo	892 ± 51[5]	−83 ± 40	1136 ± 63	68.2 ± 54		
9 mo	869 ± 49[5]	−108 ± 50	1132 ± 63	64.6 ± 54		
12 mo	873 ± 51[5]	−85 ± 41	1073 ± 58	5.3 ± 53		
Energy density of food only (kcal/g)					Group × time[2]	0.019
Baseline	1.85 ± 0.07[3,4]	—	1.74 ± 0.06[4]	—		
1 mo	1.47 ± 0.06[5]	−0.38 ± 0.09	1.17 ± 0.06	−0.57 ± 0.06		
2 mo	1.47 ± 0.06[5]	−0.37 ± 0.09	1.26 ± 0.07	−0.48 ± 0.07		
3 mo	1.41 ± 0.05[5]	−0.43 ± 0.08	1.19 ± 0.05	−0.55 ± 0.05		
6 mo	1.41 ± 0.06	−0.44 ± 0.07	1.25 ± 0.07	−0.49 ± 0.07		
9 mo	1.49 ± 0.05[5]	−0.35 ± 0.07	1.20 ± 0.04	−0.54 ± 0.06		
12 mo	1.49 ± 0.07	−0.36 ± 0.08	1.33 ± 0.04	−0.41 ± 0.06		
Fat intake (g/d)					Group[2]	0.50
					Time	<0.0001
Baseline	68.9 ± 3.3[3,4]	—	77.4 ± 4.1[4]	—		
1 mo	46.3 ± 3.4	−22.6 ± 5.2	42.6 ± 2.7	−34.8 ± 4.3		
2 mo	43.8 ± 3.1	−25.1 ± 4.7	43.5 ± 3.1	−33.9 ± 4.4		
3 mo	41.5 ± 3.2	−27.5 ± 5.0	42.9 ± 2.6	−34.5 ± 4.5		
6 mo	39.4 ± 2.2	−29.5 ± 3.5	48.8 ± 3.7	−28.6 ± 4.4		
9 mo	42.7 ± 2.8	−26.2 ± 4.1	45.6 ± 3.0	−31.8 ± 4.8		
12 mo	43.7 ± 2.8	−25.5 ± 3.9	47.9 ± 2.8	−29.5 ± 5.1		
Fruit and vegetable intake (g/d)					Group × time[2]	0.037
Baseline	277.1 ± 22.0[3]	—	364.7 ± 27.6	—		
1 mo	345.6 ± 23.7[5,6]	68.5 ± 22.4	502.8 ± 41.7[6]	138.2 ± 38.5		
2 mo	315.5 ± 26.2[5,6]	38.4 ± 27.2	464.0 ± 49.1[6]	99.3 ± 44.8		
3 mo	327.3 ± 27.8[5,6]	50.2 ± 28.6	457.0 ± 34.8[6]	92.4 ± 34.6		
6 mo	286.3 ± 23.1[5]	9.2 ± 26.7	454.8 ± 40.4	90.2 ± 36.1		
9 mo	273.9 ± 22.8[5]	−3.2 ± 22.2	446.2 ± 33.8	81.5 ± 30.4		
12 mo	302.3 ± 25.5	26.8 ± 24.1	416.9 ± 29.3	52.3 ± 32.1		
Dietary fiber intake (g/1000 kcal)					Group × time[2]	0.001
Baseline	8.4 ± 0.6[3,4]	—	9.0 ± 0.5[4]	—		
1 mo	10.4 ± 0.5[5]	2.0 ± 0.7	13.3 ± 0.8	4.3 ± 0.6		
2 mo	10.5 ± 0.5[5]	2.0 ± 0.7	13.3 ± 0.8	4.3 ± 0.8		
3 mo	10.4 ± 0.5	2.0 ± 0.6	12.0 ± 0.6	3.0 ± 0.7		
6 mo	10.9 ± 0.5	2.5 ± 0.6	11.7 ± 0.7	2.7 ± 0.7		
9 mo	10.7 ± 0.6	2.2 ± 0.6	12.1 ± 0.5	3.1 ± 0.5		
12 mo	10.9 ± 0.8	2.4 ± 0.6	12.0 ± 0.7	3.1 ± 0.7		
Physical activity (steps/d)					Group[7]	0.50
					Time	<0.0001
Baseline	5984 ± 503[3,4]	—	6731 ± 585[4]	—		
1 mo	7855 ± 503	1871 ± 584	8788 ± 413	2057 ± 532		
2 mo	8913 ± 382	2929 ± 526	8937 ± 490	2206 ± 681		
3 mo	8845 ± 398	2861 ± 553	9253 ± 528	2522 ± 659		
6 mo	8715 ± 427	2731 ± 609	8812 ± 504	2081 ± 720		
9 mo	8467 ± 498	2483 ± 604	8348 ± 490	1726 ± 698		
12 mo	9210 ± 426	3226 ± 573	9556 ± 453	2758 ± 635		

[1] All values are $\bar{x}$ ± SEM. RF, reduced-fat intervention; RF + FV, reduced-fat plus increased fruit and vegetable intervention.

[2] Differences in outcome between groups and over time were determined from a mixed linear model with repeated measures that included all available time points and that was controlled for baseline values. Unless indicated, the group × time interaction was not significant.

[3] Baseline values did not differ significantly between groups (unpaired t test).

[4] Baseline values differed significantly from those at all intervention time points ($P \leq 0.0001$), as indicated by the main effect of time in a mixed model with repeated measures.

[5] Time points at which values differed significantly between the groups with a significant group × time interaction.

[6] Values at indicated time points differed significantly from those at baseline ($P \leq 0.0005$), as indicated by the main effect of time in a mixed model with repeated measures.

[7] Random coefficients analysis was used to model the outcome across the 39 time points with control for baseline values. The group × time interaction was not significant.

$P < 0.0001$), whereas scores for disinhibition and hunger decreased at these time points (mean scores: 7.3 ± 0.3 and 4.4 ± 0.3, respectively; $P < 0.0001$). There were significant improvements in depression scores for subjects in both groups (mean score: 4.6 ± 0.4; $P < 0.0001$), as well as a significant decrease in scores for degree of binge-eating problems at both 6 and 12 mo (mean score: 7.7 ± 0.4; $P < 0.0001$).

Laboratory outcomes and blood pressure

Subjects showed beneficial changes from baseline in several physiologic values, even though baseline values were within normal ranges. Significant changes from baseline were seen in both groups for insulin, and at some time points for all cholesterol measures except LDL cholesterol (**Table 4**). Improvements in physiologic measures were significantly greater in the RF+FV group than in the RF group at various time points during the study for insulin, non-HDL cholesterol, and triacylglycerols (all $P <$ 0.044; Table 4). For LDL cholesterol and HDL cholesterol, no significant differences were found between the groups.

There were increases in both groups over 1 y in serum α-carotene, lycopene, and β-cryptoxanthin concentrations (all $P < 0.03$). The RF+FV group showed a significantly greater increase during the trial in α-carotene than did the RF group (54% increase versus 37% increase; $P = 0.023$). For lutein and zeaxanthin concentrations, there was a significant change between groups over time; subjects in the RF+FV group had greater concentrations of lutein and zeaxanthin than did subjects in the RF group at 6 mo only ($P = 0.047$).

For all subjects, there was a significant decrease from baseline in systolic and diastolic blood pressure (Table 4; $P < 0.001$); however, there were no significant differences between the groups in the blood pressure measures over time (both $P > 0.85$).

Factors predicting weight loss

To investigate which factors were most predictive of weight loss, stepwise regression analyses were performed by using data from dietary measures (energy intake, energy density of food, total fat intake, combined fruit and vegetable intake, and total weight of food), physical activity, and scores from the Eating Inventory. The primary predictor of weight loss at month 1 was the energy density of food ($R^2 = 0.16$) along with step counts ($R^2 = 0.05$; both $P < 0.040$). Together, these factors accounted for 21% of the variability in weight loss. Energy density was the only significant predictor of weight loss at month 2 ($R^2 = 0.07$; $P = 0.022$) and also at month 3 ($R^2 = 0.17$; $P = 0.0004$). The strength of the relation (regression coefficient) between food energy density and weight loss increased significantly from month 1 to month 3 ($P = 0.025$).

At months 6, 9, and 12, fruit and vegetable intake became the primary predictor of weight loss from baseline ($R^2 = 0.26$, 0.16, and 0.20, respectively; all $P < 0.0006$). The strength of the relation (regression coefficient) between fruit and vegetable intake and weight loss did not vary significantly from month 6 to month 12 ($P = 0.92$). Secondarily, the increase in the Eating Inventory dietary restraint score and the decrease in the Eating Inventory hunger score predicted weight loss during the second phase of the study (all $P < 0.029$). These scores predicted an additional 20% of the variability in weight loss at month 6 and an additional 11% at month 12.

DISCUSSION

In this randomized controlled trial with obese women, advice to reduce dietary energy density led to significant weight loss, most of which was maintained over 1 y. Participants in both intervention groups lost weight without being given specific limits for daily intakes of energy or fat, and weight loss led to beneficial changes in serum lipids and plasma insulin. The magnitude of weight lost was significantly affected by the dietary strategy that the participants were instructed to follow. Participants who were advised to decrease the energy density of their diets by adding water-rich foods and reducing their fat intake lost 33% more weight at 6 mo than did those who were advised to decrease their fat intake alone. The 2 strategies were similar in effectiveness for maintaining weight during the second 6 mo of the study. Thus, advice to reduce the energy density of the diet, especially by combining fat reduction with increased consumption of fruit and vegetables, was effective for weight loss and maintenance.

Analysis of diet records indicated that both strategies resulted in a decrease in dietary energy density. By following the same dietary advice for fat reduction, participants in both groups decreased their reported consumption of fat from 34% to 28% of energy. Participants who received additional advice to consume more water-rich foods increased their reported fruit and vegetable intake, which contributed to a further reduction in energy density. Over the course of the intervention, subjects in this group also reported eating 25% more food by weight and rated hunger significantly lower than did subjects who were advised only to reduce their fat intake. It is probable that greater food intake and reduced hunger led to better adherence to the diet, thus contributing to the greater weight loss.

Previous clinical trials found that advice to reduce dietary fat was associated with decreased energy intake and body weight (52, 53). The results of the present study provide further evidence of the effectiveness of fat reduction for weight loss. In previous trials of dietary fat reduction, however, dietary energy density was not assessed. It is important to evaluate the impact of fat reduction on energy density because these 2 dietary factors can change independently. Several shorter-term studies indicated that the effect of fat reduction on energy intake is mediated by a reduction in energy density (54). The present trial further suggests that fat reduction leads to weight loss by contributing to a reduction in the energy density of the diet. Regression analysis showed that dietary energy density was the main predictor of weight loss during the period of greatest weight change, and that dietary fat intake did not independently predict weight loss after energy density was accounted for. It is critical that future studies of the effects of macronutrients on body weight evaluate the contribution of energy density to the outcome.

Although fat reduction is one strategy for reducing dietary energy density, the addition of water-rich foods can further reduce energy density. In the present trial, advice to incorporate water-rich foods into a reduced-fat diet was more effective in controlling hunger, reducing body weight, and improving some physiologic measures than was simply following a reduced-fat diet. In addition to these outcomes, there are several other advantages of advice to increase consumption of water-rich foods. Such advice presents a positive message that balances the restrictive message to consume less fat; it has been shown that using positive messages results in greater dietary changes (55).

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TABLE 4
Insulin, lipid, and blood pressure measures in study completers over the course of 1 y[1]

	RF group (n = 36)		RF + FV group (n = 35)		Difference between groups	
Outcome	Value	Change from baseline value	Value	Change from baseline value	Effect	P
Insulin (μU/mL)					Group × time[2]	0.016
Baseline	20.3 ± 1.0[3]		20.9 ± 1.2			
3 mo	17.6 ± 1.0[4,5]	−2.7 ± 0.8	15.9 ± 0.8[4]	−5.1 ± 0.8		
6 mo	17.8 ± 0.9[4]	−2.6 ± 0.9	18.6 ± 1.2[4]	−2.4 ± 0.9		
12 mo	17.7 ± 1.3[4]	−2.8 ± 1.1	17.4 ± 1.0[4]	−3.6 ± 1.1		
Total cholesterol (mg/dL)					Group[2]	0.055
					Time	0.024
Baseline	201.7 ± 5.6[3]		197.0 ± 5.1			
3 mo	203.1 ± 6.8	+3.3 ± 4.2	187.2 ± 6.0	−9.8 ± 4.2		
6 mo	196.2 ± 6.1[4]	−4.1 ± 3.8	187.0 ± 5.5[4]	−10.0 ± 3.8		
12 mo	206.9 ± 6.8	+5.5 ± 4.6	194.2 ± 6.9	−3.8 ± 4.7		
LDL cholesterol (mg/dL)					Group[2]	0.12
					Time	0.16
Baseline	119.7 ± 4.4[3]		122.1 ± 4.4			
3 mo	117.0 ± 4.8	−0.2 ± 4.0	116.5 ± 5.2	−5.6 ± 3.9		
6 mo	117.2 ± 5.3	−0.7 ± 3.4	116.1 ± 4.6	−6.0 ± 3.3		
12 mo	127.7 ± 5.7	+7.4 ± 4.0	119.2 ± 5.4	−4.2 ± 3.9		
HDL cholesterol (mg/dL)					Group[2]	0.09
					Time	0.004
Baseline	52.7 ± 2.6[3]		47.1 ± 1.7			
3 mo	49.5 ± 2.2[4]	−3.3 ± 1.4	46.0 ± 1.8[4]	−1.1 ± 0.7		
6 mo	52.6 ± 2.3	−0.3 ± 1.3	48.8 ± 1.7	+1.7 ± 0.8		
12 mo	54.1 ± 2.8[4]	+1.3 ± 1.3	51.1 ± 2.2[4]	+3.6 ± 0.9		
Non-HDL cholesterol (mg/dL)					Group × time[2]	0.043
Baseline	149.0 ± 5.2[3]		149.9 ± 5.5			
3 mo	153.6 ± 6.7[5]	6.6 ± 4.0	141.2 ± 5.9	−8.7 ± 4.8		
6 mo	143.6 ± 5.9[4]	−3.7 ± 3.6	138.2 ± 5.5[4]	−11.7 ± 3.8		
12 mo	152.8 ± 6.2[5]	3.8 ± 5.0	143.1 ± 6.9	−7.8 ± 4.3		
HDL:total cholesterol					Group[2]	0.044
					Time	0.022
Baseline	4.1 ± 0.2[3]		4.4 ± 0.2			
3 mo	4.4 ± 0.2	+0.4 ± 0.1	4.2 ± 0.2	−0.1 ± 0.2		
6 mo	3.9 ± 0.2[4]	−0.1 ± 0.1	4.0 ± 0.2[4]	−0.4 ± 0.1		
12 mo	4.1 ± 0.2	−0.01 ± 0.1	4.0 ± 0.2	−0.4 ± 0.1		
Triacylglycerols (mg/dL)					Group × time[2]	0.017
Baseline	149.1 ± 13.4[3]		139.1 ± 11.2			
3 mo	184.0 ± 22.3[4,5]	+38.3 ± 12.1	123.9 ± 7.9	−15.1 ± 12.1		
6 mo	140.6 ± 15.9[5]	−6.1 ± 9.5	111.2 ± 7.9[4]	−27.9 ± 9.5		
12 mo	135.8 ± 14.0	−13.3 ± 11.7	119.6 ± 12.0	−17.0 ± 11.8		
Systolic blood pressure (mm Hg)					Group[6]	0.85
					Time	0.004
Baseline	119.9 ± 1.7[3]		120.2 ± 1.7			
3 mo	108.6 ± 1.9[4]	−11.3 ± 1.6	110.1 ± 2.0[4]	−10.1 ± 1.3		
6 mo	112.5 ± 2.0[4]	−7.4 ± 1.7	113.6 ± 2.0[4]	−6.4 ± 1.6		
12 mo	112.8 ± 2.0[4]	−7.0 ± 1.8	113.6 ± 2.1[4]	−6.6 ± 1.4		
Diastolic blood pressure (mm Hg)					Group[6]	0.96
					Time	<0.0001
Baseline	78.4 ± 1.1[3]		79.3 ± 1.0			
3 mo	71.5 ± 1.1[4]	−6.9 ± 1.1	72.3 ± 1.2[4]	−7.0 ± 1.1		
6 mo	72.4 ± 1.5[4]	−5.9 ± 1.3	73.5 ± 1.2[4]	−5.6 ± 1.0		
12 mo	70.3 ± 1.5[4]	−8.0 ± 1.3	72.7 ± 1.4[4]	−6.6 ± 1.3		

[1] All values are $\bar{x}$ ± SEM. RF, reduced-fat intervention; RF + FV, reduced-fat plus increased fruit and vegetable intervention.
[2] Differences in outcome between groups and over time were determined from a mixed linear model with repeated measures that included all available time points and that was controlled for baseline values. Unless indicated, the group × time interaction was not significant.
[3] Baseline values did not differ significantly between groups (unpaired t test).
[4] Values at indicated time points differed significantly from those at baseline ($P \leq 0.001$), as indicated by the main effect of time in a mixed model with repeated measures.
[5] Time points at which values differed significantly between the groups with a significant group × time interaction.
[6] Random coefficients analysis was used to model the outcome across the 39 time points with control for baseline values. The group × time interaction was not significant.

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Increasing the consumption of fruit and vegetables is also consistent with current dietary guidelines and is associated with high diet quality (56). Promoting increased fruit and vegetable intake for weight management may lead to beneficial effects on other health-related outcomes, such as reduction of risk for chronic illness (57–60).

Although there are many advantages to fruit and vegetable consumption, factors such as cost, convenience, and preparation time are often cited as barriers to increased intake (61). In this trial, although subjects in the 2 groups differed in reported consumption of fruit and vegetables, they gave similar ratings of satisfaction with the 2 dietary approaches, particularly for cost, convenience, and food preparation. These data indicate that specific instruction in techniques for increasing fruit and vegetable intake was both effective and well accepted by the participants.

In addition to the strategies to reduce dietary energy density, the effectiveness of the weight-loss interventions was enhanced by the physical activity component. Participants in both groups exceeded their goal of an additional 2000 steps/d and increased their daily step counts by an average of 38%. Subjects maintained a significant increase in step counts over the course of the intervention, which confirms continued adherence to the program for 1 y. These sustained behavioral changes are likely to have contributed to the maintenance of weight loss (62–64).

The detailed diet records kept by the participants showed that energy intake during the intervention declined significantly from baseline values in both groups. Between the groups, however, there were no significant differences in reported energy intake that corresponded with the differences in weight loss. This may be explained by the large variability in daily energy intake, which limited the statistical power to detect modest differences in intake given the number of subjects in the trial. Conversely, the much larger differences between groups in the weight of food consumed allowed for the detection of significant changes in both food weight and dietary energy density. In self-reported diet records from obese subjects there are also issues regarding underreporting of intake (65) and compliance bias (66, 67). Because all participants in this trial were obese and received the same amount of dietary information and counseling, the potential for underreporting and bias was likely to have been similar in both groups. The advice given to one group to increase intake of water-rich foods may have influenced those participants to report a greater intake than they were actually consuming. It is unlikely that compliance bias fully explains the reported increase in fruit and vegetable consumption in this group, however, because the increase corresponded with an increase in plasma carotenoids.

The promising results of this trial indicate the need to investigate the effects of low-energy-density diets in a larger multicenter trial, which could include a diversity of populations and a variety of settings. Future studies should also investigate the effectiveness of a less intensive intervention. Adherence to both the diet and physical activity advice may have been improved by the frequent contact with dietitians, which has been reported to increase compliance in other studies (68, 69). Of additional interest is determining whether adherence to a diet low in energy density persists beyond 1 y. Clinical data from a weight-loss program in which individuals were advised to add low-energy-density foods to a reduced-fat, energy-restricted diet showed that weight loss was well maintained for 2 to 6 y after treatment (70, 71), and at 2 y maintenance was associated with a low-energy-density dietary pattern (72).

The 2 strategies for reducing energy density that were tested in this trial were both effective in reducing body weight and maintaining weight loss without prescribing limits for energy or fat intake. Diets that are low in energy density, particularly those that encourage increased consumption of water-rich foods, allow individuals to eat adequate amounts of food while restricting energy intake, which helps to control hunger. Because a wide variety of foods can be included in a diet that is reduced in energy density (56, 73), this type of eating plan can be adapted to individual food preferences. Individuals should be encouraged to find a low-energy-density eating pattern that is nutritionally balanced and that can be sustained for lifelong weight management.

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The contributions of the authors were as follows—JAE-M, LSR, and BJR: study concept and design; JAE-M and AMB: acquisition of data; JAE-M, LSR, JHL, and BJR: analysis and interpretation of data; JAE-M, LSR, and BJR: drafting of the manuscript; LSR, JHL, and BJR: critical revision of the manuscript; and JAE-M, AMB, and BJR: study supervision. None of the authors had any conflicts of interest.

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Exhibit F

Effects of a reduced-glycemic-load diet on body weight, body composition, and cardiovascular disease risk markers in overweight and obese adults[1–3]

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ABSTRACT

Background: Lowering the dietary glycemic load and increasing protein intake may be advantageous for weight management.

Objective: This randomized controlled trial was designed to evaluate the effects of an ad libitum reduced-glycemic-load (RGL) diet on body weight, body composition, and cardiovascular disease (CVD) risk markers in overweight and obese adults during an initial weight-loss phase (12 wk) and a weight-loss maintenance phase (weeks 24–36).

Design: Subjects were assigned to RGL ($n = 43$) or low-fat, portion-controlled (control; $n = 43$) diet groups. The RGL group was instructed to eat until satisfied, maintaining a low carbohydrate intake during weeks 0–2 and adding low-glycemic-index carbohydrate thereafter. Control subjects were instructed to reduce fat intake and decrease portion sizes, with a targeted energy deficit of 500 to 800 kcal/d.

Results: The RGL group had lost significantly more weight than did the control group at week 12 (−4.9 and −2.5 kg, respectively; $P = 0.002$), but the 2 groups did not differ significantly at week 36 (−4.5 and −2.6 kg, respectively; $P = 0.085$). Changes in fat mass differed between the groups at week 12 (−1.9 and −0.9 kg, respectively; $P = 0.016$) but not at week 36 (−2.0 and −1.3 kg, respectively; $P = 0.333$). At the end of the study, no differences were found in responses for CVD risk markers except a larger mean change in HDL cholesterol in the RGL group than in the control group (3.8 and 1.9 mg/dL, respectively; $P = 0.037$).

Conclusion: These findings provide evidence that an ad libitum RGL diet is a reasonable alternative to a low-fat, portion-controlled eating plan for weight management. *Am J Clin Nutr* 2007;85:724–34.

KEY WORDS Glycemic load, obesity, weight loss, body composition, cardiovascular disease risk markers, glucose tolerance, randomized controlled trial

INTRODUCTION

The prevalence of obesity in the United States has more than doubled during the past 25 y (1). Recent estimates from population-based samples suggest that nearly two-thirds of adults in the United States are overweight or obese (2). This fact is generating considerable public health concern because excess adiposity is associated with a greater risk of the development of diabetes mellitus, atherosclerotic cardiovascular disease (CVD), and several forms of cancer (1).

Traditional approaches to weight management have emphasized restricting dietary fat, replacing fats with carbohydrates, and limiting portion sizes. Long-term compliance with such diets has proven difficult, in part because physiologic adaptations during energy restriction appear to increase hunger and reduce energy expenditure (EE) (3–5).

Recently, it was suggested that lowering the dietary glycemic load (GL) through a combination of moderate carbohydrate restriction, consumption of slowly digested and absorbed forms of dietary carbohydrate [ie, low-glycemic-index (low-GI) foods], or both may be advantageous for persons who are attempting to lose excess body weight (3, 5, 6). In settings in which food intake could be closely controlled, low-GL, energy-restricted diets were associated with smaller declines in resting EE, lower hunger ratings, and greater improvements in several risk markers for CVD than were traditional low-fat, energy-restricted diets (3, 5, 7).

In several trials, free-living subjects also showed greater weight loss over 3–6 mo with reduced-carbohydrate or low-GL diets than with traditional low-fat, portion-controlled diets (8–13), whereas others showed no clear advantage from low-carbohydrate or low-GI diets (14–17). In longer trials, differences in weight loss between low-fat and low-GL diet groups after 1 y were small (9, 15, 17–19). However, some investigators noted favorable differences in fasting triacylglycerol (9, 17, 18) and HDL-cholesterol (9, 18) concentrations and in glycosylated hemoglobin in the subset of those with diabetes mellitus (18).

The current randomized trial was designed to assess the effects of an ad libitum RGL diet that was intended to include a moderate intake of dietary carbohydrate with emphasis on low-GI foods and lean sources of protein to replace some high-carbohydrate foods. This approach was compared with a traditional, low-fat, portion-controlled dietary program in free-living, overweight and obese men and women. The primary outcomes were changes in body weight from baseline to the end of an initial 12-wk

[1] From Radiant Research, Chicago, IL (KCM, VNK, and MHD); Provident Clinical Research, Bloomington, IN (KCM); and Kraft Foods, Inc, Glenview, IL (TMR and KRR).

[2] Supported by Kraft Foods.

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weight-loss period and at the end of a weight-maintenance period at week 36. Secondary outcomes included changes in body composition, selected CVD risk markers, and health-related quality of life at weeks 12 and 36.

SUBJECTS AND METHODS

Subjects

Potential participants were recruited from the Chicago metropolitan area and screened by telephone. Eligibility was further assessed at a screening visit. To qualify for entry into the study, men and women had to be aged 18–65 y, have a waist circumference measurement at week −1 of ≥87 cm for women and ≥90 cm for men, and be judged by the investigators to be in good health on the basis of medical history and routine laboratory tests. Subjects had to be willing to discontinue all use of dietary supplements or multivitamins, except those provided during the study, and to follow the assigned diet and maintain their usual level of physical activity throughout the trial.

Volunteers were excluded from participation if they had experienced a weight loss of >4.5 kg in the 2 mo before screening, had a body mass index (BMI; in kg/m^2) ≥37.0, were current smokers (any cigarette use), or had a history of smoking in the 6 mo before screening. Subjects were also excluded from participation if they had diabetes mellitus, uncontrolled hypertension, or a history of cancer (other than successfully resected basal cell carcinoma) in the past 2 y.

Subjects with a history of or current significant cardiac, renal, pulmonary, hepatic, biliary, or endocrine disease were excluded, as were those with a history of recurrent nephrolithiasis or acute nephrolithiasis within the year before screening. Subjects were excluded from participation if they had used any weight-loss medication, supplements, programs, or meal-replacement products intended to alter body weight during the 4 wk before screening or if they had any diagnosed eating disorder, a history of surgery for weight-reducing purposes, or a clinically significant gastrointestinal disorder.

Additional exclusion criteria included the use of systemic corticosteroids, androgens, phenytoin, or pseudoephedrine; lipid-lowering therapies (unless dose-stable for 2 mo before enrollment); drugs for regulating hemostasis other than dose-stable aspirin; thyroid hormones (except stable-dose replacement therapy for ≥2 mo before enrollment); or psychiatric medications.

Postmenopausal women who were current users of sex hormone therapy or who had discontinued use in the 2 mo before screening were excluded. Female subjects who were pregnant, planning to be pregnant during the study period, or lactating or those of childbearing potential who were not using an approved method of contraception were also excluded.

This trial was performed according to Good Clinical Practice Guidelines, the Declaration of Helsinki (2000), and US 21 CFR Part 50 – Protection of Human Subjects, and Part 56 – Institutional Review Boards. An institutional review board, Quorum (Seattle, WA), approved the protocol and the informed consent document before the initiation of the study. Study procedures were reviewed with subjects, and each participant provided written informed consent and authorization for the release of protected health information before study procedures were carried out.

Clinic visits

This trial used a randomized, controlled design with 2 parallel treatment arms. Subjects were randomly assigned to either the RGL or portion-controlled (control) diet. Weeks 0–12 were weight-loss treatment. At some point between weeks 12 and 24, each subject transitioned to a weight-maintenance phase. From week 24 on, all subjects were in the weight-maintenance phase.

The study included 15 clinic visits: 1 screening visit (week −1), 1 visit at baseline (week 0), 7 visits during the weight-loss treatment phase (weeks 1, 2, 4, 6, 8, 10, and 12), 3 visits during the transition from weight-loss treatment to weight-loss maintenance (weeks 16, 20, and 24), and 3 visits during the weight-loss maintenance phase (weeks 28, 32, and 36).

Visits were conducted between 0700 am and 1200. Subjects were asked to refrain from consuming any foods or beverages except water for ≥10 h and to abstain from consuming alcohol for ≥24 h before visits.

At week −1, subjects provided written informed consent and completed a medical history questionnaire. Concomitant medications used 8 wk before and during the study were recorded at this and subsequent visits. Body weight, height, and waist circumference were measured, and vital signs were assessed. Samples were taken for serum chemistry, hematology, lipid panel, and urinalysis.

At the randomization (baseline) visit (week 0), subjects received diet instruction and information on their assigned diets for them to take home. All subjects were provided with daily multivitamins (Centrum; Wyeth Consumer Healthcare, Madison, NJ) and were instructed to bring back any unused multivitamins at each subsequent study visit.

At each visit, vital signs were assessed and anthropometric measurements were taken, daily food checklists were reviewed and used in dietary counseling or reinforcement, and any concomitant use of medication and adverse events were assessed. Fasting serum lipid values were obtained at weeks −1, 0, 10, 12, 32, and 36. Serum chemistry, hematology, urinalysis, and fasting insulin and glucose values were measured at weeks −1, 12, and 36. Dual-energy X-ray absorptiometry (DXA) was conducted at weeks 0, 12, 24, and 36 for assessment of body composition. Three-day diet records were analyzed by using the NUTRIENT DATA SYSTEM FOR RESEARCH (version 4.06; University of Minnesota, Minneapolis, MN) at weeks 0, 2, 6, 12, 24, and 36. The Willett Food-Frequency Questionnaire was completed at weeks 0, 12, and 36 for estimation of dietary GI and GL (20). The questionnaire uses published sources of GI values for carbohydrates from specific foods for calculation of the weighted average GI of the diet (20). GL is calculated as the weighted average GI multiplied by the total daily carbohydrate intake (20).

Laboratory measurements

The dipstick test (Ketostix; Bayer Diagnostics, Tarrytown, NY) was used to assess urinary ketone concentrations. Other laboratory measurements, including serum chemistry, hematology, insulin, urinalysis, and lipid concentrations, were conducted by Medical Research Laboratories (MRL, Highland Heights, KY). Serum chemistry analysis (including fasting glucose) was completed with a chemistry analyzer (Hitachi 747–200; Roche Diagnostics Corporation, Indianapolis, IN), and hematologic testing was conducted with the use of a complete count analyzer (Coulter STKS; Coulter Corporation, Miami, FL).

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Cholesterol and triacylglycerol were measured enzymatically by using the Hitachi 747–200. Heparin and manganese chloride were used to precipitate apolipoprotein-containing particles to allow measurement of HDL cholesterol. LDL cholesterol in mg/dL was calculated by using the Friedewald equation: LDL cholesterol = (total cholesterol – HDL cholesterol – triacylglycerol)/5 (21). The homeostasis model assessment (HOMA) of insulin resistance was calculated as glucose (mmol/L) × insulin (mU/L)/22.5 (22).

Blood pressure

Blood pressure was obtained after the subject had been sitting quietly for 5 min. Systolic and diastolic pressures were measured with a standard manual mercury sphygmomanometer. Two measurements, separated by 2 min, were taken and averaged. If the measurements differed by >5 mm Hg, an additional reading was obtained, and all 3 readings were averaged.

Body composition and anthropometric measurements

Whole-body DXA scans were performed (QDR 4500A; Hologic Inc, Waltham, MA). Body composition values (fat mass and fat-free mass) were measured with the use of HOLOGIC SYSTEMS Software (version 9.03D; Hologic Inc) according to the procedures outlined in the Hologic QDR 4500 user's guide of 1995.

Anthropometric measurements included subjects' height (first visit only), weight, and waist circumference. At each visit, the measurement of waist circumference was performed at the level of the iliac crest by using a nonstretchy anthropometric tape measure. Two measurements were taken and averaged. If these differed by >0.5 cm, a third measurement was taken and the outlying value was discarded.

Diet implementation

Diet counselors were the same for both groups. Diet training manuals were created to standardize training of the counselors, all of whom were Registered Dietitians with extensive experience in counseling for weight management. For both diet groups, handouts were provided to each subject for at-home use, and dietary guidance was reinforced at each treatment visit.

For subjects assigned to the control diet, energy needs for weight maintenance were estimated from basal EE calculated with the Harris-Benedict equation (23) multiplied by an activity factor of 1.2, 1.3, or 1.4 after evaluation of activity level from the results of the Stanford 7-d physical activity questionnaire (24). On the basis of a review of diet records returned, recommendations were made on substitutions for high-fat foods and decreasing portion sizes and energy density to produce a target energy deficit of 500 to 800 kcal/d, while consuming a nutritionally balanced diet. Subjects were encouraged to achieve weight loss of ≈0.5 kg/wk.

Those following the RGL diet were instructed to eat 3 meals/d plus snacks and to eat until hunger was satisfied. Specific high-carbohydrate foods, including all starchy foods and fruits, were eliminated during phase 1 of the diet (the first 2 wk). Subjects were advised not to consume any alcohol during this period and were instructed on selection of foods with low carbohydrate content that are not high in saturated and *trans* fats to avoid excessive intake of cholesterol-raising fatty acids. At week 2, subjects received instructions for the second phase of the diet (weeks 2–12), during which low-GI foods, such as high-fiber fruits and vegetables, beans, and whole-grain breads and cereals, were restored to the diet. Moderate alcohol consumption was allowed after week 2.

At week 12, both groups were given the option to remain in the weight-loss phase of the diet or switch to the weight-loss maintenance phase. Subjects who opted to continue in the weight-loss phase at week 12 were given the option to switch at each subsequent visit until week 24 to accommodate individual weight-loss goals. At week 24, all subjects who had not yet done so were asked to switch to weight-loss maintenance. During the weight-maintenance phase, subjects were advised to continue the portion-controlled or RGL diet while eating sufficient quantities of food to maintain the level of body weight achieved at the time of switching from weight loss to weight maintenance.

Assessment of quality of life

A quality-of-life instrument was completed by all subjects at weeks 0, 12, 24, and 36 (SF-36 Health Survey 2002; Medical Outcomes Trust and QualityMetric Inc, Lincoln, RI).

Statistical analysis

Statistical analyses were conducted with SAS software (versions 8.02 and 9.1; SAS Institute, Cary, NC). All tests of statistical significance were performed at alpha = 0.05, 2-sided, unless otherwise indicated. The Shapiro-Wilk test (25) was used to test variables for normality. Where necessary because of nonnormality, rank transformations were employed before calculation of inferential statistics. Baseline comparability of treatment groups was assessed by 1-factor analysis of variance (ANOVA) for continuous variables and chi-square or Fisher's exact test for categorical variables.

An intent-to-treat analysis of outcome variables was completed by using all data for subjects with ≥1 postrandomization body weight measurement. For this analysis, which was considered primary, the last nonbaseline observation was carried forward for missing data points. A secondary analysis for body weight was also completed, in which the baseline weight was substituted after discontinuation for all subjects who dropped out during the treatment period. In addition, analyses were completed that excluded subjects who did not complete the 36-wk study or who violated the protocol in some material way. However, because results from these analyses did not differ materially from the results from the intent-to-treat analyses, only the latter are presented.

For all continuous variables, repeated-measures ANOVA models were employed for values at baseline and weeks 2 (dietary variables only), 12, and 36. These models each included terms for treatment, time, and treatment × time interaction. Pairwise comparisons between groups were completed for individual timepoints when the treatment × time interaction term was significant ($P \leq 0.05$) in the repeated-measures model. Body weight responses at weeks 12 and 36 were considered the primary outcome variables and were assessed by ANOVA, with baseline body weight and treatment group as factors in the model. For both the primary and secondary body weight response analyses, the P values obtained at weeks 12 and 36 were adjusted (multiplied by 1.724) to account for the 2 comparisons (26). This adjustment equated to the requirement of a nominal, 2-sided P value of 0.029 to obtain statistical significance.

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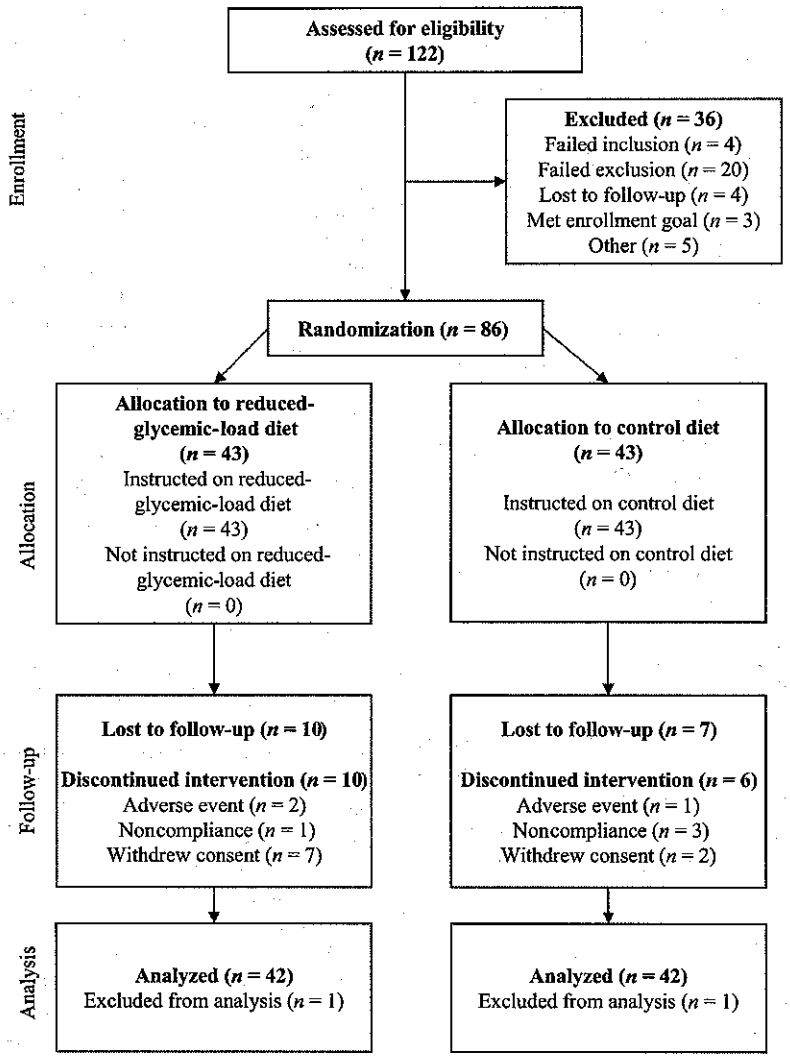

FIGURE 1. Subject disposition by treatment group assignment.

In addition, previously specified analyses of changes from baseline to week 12 (end of the initial weight-loss period) and week 36 (end of the weight-maintenance period) were completed for body composition, anthropometric, and laboratory variables by using 1-factor ANOVA with treatment as a fixed effect. Exploratory analyses were completed to assess maximal weight loss and the changes from the point of maximal weight loss to week 36 in both treatment groups. Multiple linear regression analysis was used to explore determinants of the changes in body composition. For all secondary outcome and exploratory variables, unadjusted *P* values are presented.

Safety analyses included all subjects who were randomly assigned. Safety and tolerability were assessed by evaluation of adverse events that occurred during treatment, laboratory test results, and vital signs measurements. Adverse events were coded by using the World Health Organization dictionary. Fisher's exact test was used to test for differences between groups in adverse events overall and for each body system and coded term.

RESULTS

Subjects

A summary of subject disposition is shown in **Figure 1**. A total of 122 men and women were screened for entry into the trial, 86 of whom qualified and were randomly assigned ($n = 43$/group). No significant differences were observed between groups in the percentage of subjects who participated in the study through weeks 12 (81.4% in each group; $P = 1.00$) and 36 (53.5% in the RGL group and 69.8% in the control group; $P = 0.120$), respectively.

Baseline characteristics of the study sample are shown in **Table 1**. Subjects were approximately two-thirds female. Fifty-two percent classified themselves as non-Hispanic white and 35% as African American. The mean age of the participants was 50 y; mean BMI was ≈32, and 67% of the subjects were obese (BMI ≥30). The fasting insulin concentration tended to be higher among subjects in the RGL group than among those in the control group (10.3 and 9.0 mU/L, respectively; $P = 0.061$). Otherwise, the groups were well matched with regard to baseline characteristics.

TABLE 1
Baseline characteristics by treatment group[1]

Characteristic	RGL group (n = 43)	Control group (n = 43)	P[2]
Sex [n (%)]			
Women	29 (67.4)	29 (67.4)	1.000
Men	14 (32.6)	14 (32.6)	
Ethnicity [n (%)]			
Non-Hispanic white	19 (44.2)	26 (60.5)	0.129
African American	15 (34.9)	15 (34.9)	
Hispanic	6 (14.0)	1 (2.3)	
Other	3 (7.0)	1 (2.3)	
Age (y)	47.9 ± 1.8[3]	51.4 ± 1.5	0.142
Weight (kg)	91.2 ± 2.0	88.7 ± 1.8	0.353
BMI (kg/m^2)	32.1 ± 0.6	31.6 ± 0.5	0.580
Physical activity (MET-h/wk)	312.2 ± 14	309.8 ± 10.0	0.672

[1] RGL, reduced glycemic load; MET, metabolic equivalent.
[2] P for continuous variables generated from ANOVA with treatment as a factor and for categorical variables from 2 × 2 chi-square tests (ethnicity was classified as non-Hispanic white or other).
[3] $\bar{x}$ ± SEM (all such values).

One subject in each group dropped out of the trial after randomization but before providing any individual data for body weight or composition. These subjects were excluded from the intent-to-treat analyses.

Body weight and composition

At the end of the initial 12-wk weight-loss period, the mean weight change was −4.9 ± 0.5 kg in the RGL arm and −2.5 ± 0.5 kg in the control arm (adjusted P = 0.002), as shown in **Figure 2**. At week 12, 24 subjects (55%) in the RGL group and 9 subjects (21%) in the control group had achieved a loss of ≥5% of body weight (P = 0.002). An exploratory analysis showed that mean maximum weight loss was significantly (P = 0.005) larger in the RGL group (6.4 ± 0.6 kg) than in the control group (4.4 ± 0.6 kg).

By the end of the weight-maintenance period (at week 36), the mean weight change was −4.5 ± 0.7 kg in the RGL group and −2.6 ± 0.9 kg in the control group (adjusted P = 0.085), as shown in Figure 2. At week 36, 45% and 29% of subjects in the RGL and control groups, respectively, were ≥5% below their baseline body weights (P = 0.114). An exploratory analysis of weight change from the point of maximal weight loss to week 36 found that the groups did not differ significantly in the mean amount of weight regained during the weight-maintenance period (1.9 ± 0.3 kg in the RGL group and 1.8 ± 0.3 kg in the control group; P = 0.866).

In the secondary analysis, in which the baseline body weight was substituted for missing values after subject dropout, the change from baseline to week 12 differed between groups, with significantly (P = 0.018) greater weight loss apparent in the RGL group than in the control group (−4.9 ± 0.6 and −3.0 ± 0.6 kg, respectively). However, no significant difference between groups was present at week 36 (−2.8 ± 0.7 and −2.2 ± 0.8 kg for the RGL and control groups, respectively; P = 0.684).

As shown in **Table 2**, body fat and fat-free mass were lower in both groups at week 12, and the reductions in the RGL arm were significantly (P = 0.016 and < 0.001 for body fat and fat-free mass, respectively) greater in the RGL arm than in the control arm. A significant (P = 0.004) difference between groups in the loss of fat-free mass persisted at 36 wk, whereas changes in fat mass from baseline were no longer significantly different (−2.0 and −1.3 kg for the RGL and control groups, respectively; P = 0.333). No significant difference in the loss of fat-free mass at week 12 was seen between groups after adjustment for body weight loss by using multiple linear regression (P = 0.162). However, RGL treatment was associated with significantly (P = 0.037) greater loss of fat-free mass at week 36, after adjustment for the difference in body weight response, than was the control diet. Waist circumference also declined in both groups, but the difference in response between groups was not significant at week 12 (P = 0.082) or week 36 (P = 0.783).

Physical activity and dietary composition

No significant differences between groups were observed in physical activity at baseline (Table 1) or at any timepoint during treatment (data not shown). Estimates of dietary composition

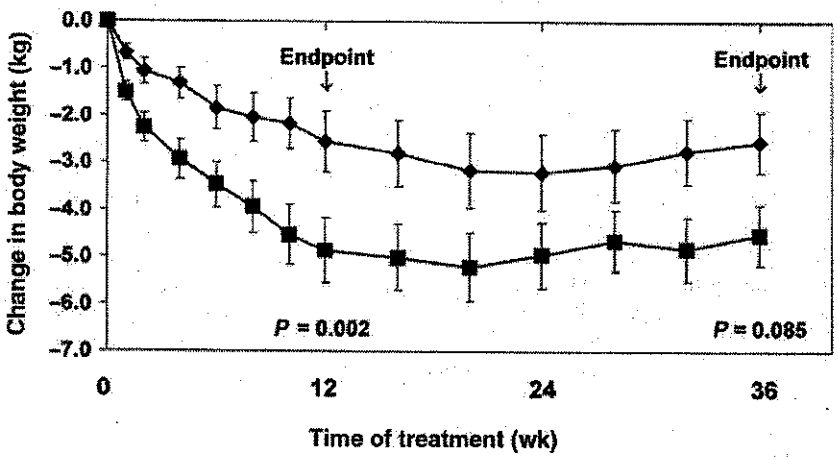

FIGURE 2. Least-squares mean (±SEM) changes in body weight from baseline by treatment group assignment: ◆, control group (n = 42); ■, reduced-glycemic-load group (n = 42). The last available nonbaseline value was carried forward to impute missing values. Significant effects were present for time and treatment × time interaction (P < 0.001 for both) in an initial repeated-measures ANOVA model. P values shown in the figure were generated from ANOVA models with baseline body weight and treatment group as factors; they were multiplied by 1.724 to adjust for 2 previously specified endpoint comparisons (weeks 12 and 36).

TABLE 2
Body composition and waist circumference values by time point and treatment group[1]

Variable	RGL group		Control group		*P*		
					Time[2]	Treatment × time[2]	Pairwise[3]
	n		*n*				
Fat mass (kg)							
Baseline	42	30.1 ± 1.1[4]	42	29.0 ± 1.0	<0.001	0.027	0.466
Δ at week 12	37	−1.9 ± 0.3	37	−0.9 ± 0.3	—	—	0.016
Δ at week 36	37	−2.0 ± 0.5	37	−1.3 ± 0.5		—	0.333
Fat-free mass (kg)							
Baseline	42	57.0 ± 1.6	42	55.6 ± 1.6	<0.001	0.004	0.552
Δ at Week 12	37	−2.2 ± 0.2	37	−1.0 ± 0.3	—	—	<0.001
Δ at Week 36	37	−2.1 ± 0.3	37	−0.9 ± 0.3	—	—	0.004
Waist circumference (cm)							
Baseline	42	105.1 ± 1.6	42	101.8 ± 1.4	<0.001	0.003	0.136
Δ at Week 12	37	−4.3 ± 0.7	37	−2.8 ± 0.5	—	—	0.082
Δ at Week 36	37	−3.1 ± 0.8	37	−2.8 ± 0.7	—	—	0.783

[1] RGL, reduced glycemic load.
[2] *P* values from repeated-measures ANOVA models with treatment, time, and treatment × time interaction as factors.
[3] *P* values from ANOVA models comparing treatments at each timepoint.
[4] $\bar{x}$ ± SEM (all such values).

from analysis of 3-d food records and Willett Food-Frequency Questionnaires (for GI and load) are shown in **Table 3**. The groups did not differ significantly at baseline in any variable except alcohol intake, which was higher in the RGL group than in the control group (6 and 2 g/d, respectively; $P = 0.010$).

Reported energy consumption was reduced from baseline in both groups throughout the treatment period. Differences between treatment arms did not reach significance, although mean values were 4.7% to 10.5% lower in the RGL group than in the control group at each timepoint after randomization. Consistent with the treatment plan, dietary carbohydrate and sugars (sucrose and fructose) were markedly reduced at week 2 in the RGL group ($P < 0.001$ versus control group). Reported intakes of carbohydrate and sugars increased above the week 2 values in the RGL group but remained substantially below those intakes in the control group (for both dietary carbohydrateand sugars, $P \leq 0.005$ at weeks 12 and 36).

Mean GI in the RGL group declined from 52 at baseline to 46 at week 12 and 48 at week 36 ($P \leq 0.001$ versus control group at both timepoints) and remained essentially unchanged from baseline in the control group (52, 51, and 51 at baseline, week 12, and week 36, respectively). The combination of lower GI and reduced carbohydrate consumption produced reductions of 43% to 49% in mean GL in the RGL group but reductions of 12% to16% in the control group ($P \leq 0.002$ at weeks 12 and 36).

Alcohol was not allowed during the first 2 wk of the RGL treatment plan, which resulted in a significant difference in intake between groups at week 2 ($P < 0.001$), but intake did not differ at weeks 12 and 36. Reported protein intake was higher in the RGL group at all timepoints during treatment, but the treatment × time interaction was not significant ($P = 0.067$). Intakes of total, saturated, and unsaturated fats were also higher in the RGL group than in the control group, but differences did not reach significance. Mean dietary cholesterol intake was higher (122–259 mg/d) in the RGL group than in the control group at all timepoints during the treatment ($P < 0.001$).

Dietary fiber, expressed in g/1000 kcal, did not differ between groups. No significant differences between groups were observed in calcium, magnesium, potassium, or sodium intakes during treatment.

Cardiovascular disease risk markers, ketones, and quality of life

Mean values for CVD risk markers by group at baseline and changes from baseline are shown in **Table 4**. No significant differences between groups were present at baseline. The only significant difference in lipid responses between the RGL and control diet groups was in the increase in HDL cholesterol at week 36 (3.8 and 1.9 mg/dL, respectively; $P = 0.037$).

The RGL dietary regimen was designed to have sufficient carbohydrate to prevent significant ketonemia, even during the initial period of more severe carbohydrate restriction. At baseline, 4 subjects—1 in the RGL group and 3 in the control group—had measurable concentrations of urinary ketones ($P = 0.270$). At week 2, 12 subjects (29%) in the RGL group and 1 subject (3%) in the control group had urinary ketones ($P = 0.001$). Of the 12 subjects with urinary ketones in the RGL group, 6 were classified as having trace amounts and 6 as having small-to-moderate concentrations. At weeks 12 (14.6% in the RGL group and 7.1% in the control group; $P = 0.273$) and 36 (12.2% in the RGL group and 2.4% in the control group; $P = 0.084$), the prevalence of subjects with urinary ketones in the 2 groups did not differ significantly, but it tended to be higher in the RGL group.

No differences between groups at baseline or subsequent timepoints were observed for any domain in the SF-36 quality-of-life questionnaire (data not shown).

DISCUSSION

This randomized, controlled trial showed that an ad libitum RGL diet produced significantly greater losses of body weight and fat during an initial weight-loss period than did a traditional,

TABLE 3
Dietary composition, including dietary supplements, from analyses of 3-d diet records and Willett food-frequency questionnaires by timepoint and treatment group[1]

Variable	RGL group		Control group		*P*		
					Time[2]	Treatment × time[2]	Pairwise[3]
	n		*n*				
Energy (kcal/d)							
Baseline	42	2050 ± 131[4]	42	1961 ± 132	<0.001	0.555	0.651
Week 2	41	1365 ± 72	41	1525 ± 87	—	—	—
Week 12	41	1343 ± 54	41	1500 ± 107	—	—	—
Week 36	41	1533 ± 78	41	1608 ± 113	—	—	—
Carbohydrate (g/d)							
Baseline	42	222 ± 18	42	218 ± 17	<0.001	<0.001	0.811
Week 2	41	69 ± 6	41	168 ± 12	—	—	<0.001
Week 12	41	108 ± 6	41	171 ± 14	—	—	<0.001
Week 36	41	131 ± 9	41	186 ± 15	—	—	0.002
Sucrose + fructose (g/d)							
Baseline	42	64 ± 8	42	58 ± 6	<0.001	<0.001	0.696
Week 2	41	17 ± 2	41	44 ± 6	—	—	<0.001
Week 12	41	25 ± 3	41	46 ± 6	—	—	<0.001
Week 36	41	28 ± 2	41	51 ± 7	—	—	0.005
Glycemic index[5]							
Baseline	42	52 ± 1	42	52 ± 1	<0.001	—	0.799
Week 12	38	46 ± 1	35	51 ± 1	<0.001	—	<0.001
Week 36	38	48 ± 1	36	51 ± 1	—	—	0.001
Glycemic load[6]							
Baseline	42	14355 ± 1146	42	14409 ± 1148	<0.001	0.001	0.974
Week 12	38	7298 ± 867	35	12649 ± 1060	—	—	<0.001
Week 36	38	8173 ± 816	36	12118 ± 836	—	—	0.002
Alcohol (g/d)							
Baseline	42	6 ± 2	42	3 ± 1	0.021	0.006	0.010
Week 2	41	0 ± 0	41	3 ± 1	—	—	0.001
Week 12	41	3 ± 1	41	3 ± 1	—	—	0.907
Week 36	41	5 ± 1	41	5 ± 2	—	—	0.572
Protein (g/d)							
Baseline	42	80 ± 4	42	82 ± 5	0.065	0.067	0.832
Week 2	41	97 ± 5	41	75 ± 3	—	—	—
Week 12	41	87 ± 4	41	70 ± 3	—	—	—
Week 36	41	87 ± 4	41	76 ± 5	—	—	—
Total fat (g/d)							
Baseline	42	92 ± 7	42	85 ± 8	<0.001	0.305	0.355
Week 2	41	80 ± 6	41	62 ± 5	—	—	—
Week 12	41	63 ± 4	41	61 ± 6	—	—	—
Week 36	41	72 ± 5	41	63 ± 6	—	—	—
Saturated fat (g/d)							
Baseline	42	31 ± 3	42	28 ± 3	<0.001	0.325	0.294
Week 2	41	24 ± 2	41	19 ± 2	—	—	—
Week 12	41	20 ± 2	41	19 ± 2	—	—	—
Week 36	41	23 ± 2	41	18 ± 2	—	—	—
Unsaturated fat (g/d)							
Baseline	42	54 ± 4	42	51 ± 5	<0.001	0.327	0.552
Week 2	41	49 ± 4	41	38 ± 3	—	—	—
Week 12	41	38 ± 2	41	37 ± 3	—	—	—
Week 36	41	43 ± 3	41	40 ± 4	—	—	—
Cholesterol (mg/d)							
Baseline	42	317 ± 26	42	273 ± 24	<0.001	0.003	0.266
Week 2	41	487 ± 45	41	228 ± 19	—	—	<0.001
Week 12	41	347 ± 27	41	221 ± 20	—	—	<0.001
Week 36	41	339 ± 23	41	217 ± 20	—	—	<0.001
Dietary fiber ($g \cdot d^{-1} \cdot 1000\ kcal^{-1}$)							
Baseline	42	8 ± 1	42	9 ± 1	0.050	0.393	0.456
Week 2	41	11 ± 1	41	12 ± 1	—	—	—
Week 12	41	13 ± 1	41	12 ± 1	—	—	—
Week 36	41	11 ± 1	41	12 ± 1	—	—	—
Calcium (mg/d)							
Baseline	42	715 ± 69	42	724 ± 74	0.056	0.453	0.979
Week 2	41	886 ± 60	41	783 ± 41	—	—	—
Week 12	41	824 ± 45	41	793 ± 55	—	—	—
Week 36	41	838 ± 51	41	839 ± 54	—	—	—

(*Continued*)

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TABLE 3 (*Continued*)

Variable	RGL group		Control group		*P*		
					Time[2]	Treatment × time[2]	Pairwise[3]
	n		*n*				
Magnesium (mg/d)n							
Baseline	42	270 ± 19	42	272 ± 19	<0.001	0.207	0.583
Week 2	41	361 ± 16	41	342 ± 14	—	—	—
Week 12	41	353 ± 11	41	347 ± 18	—	—	—
Week 36	41	360 ± 14	41	376 ± 23	—	—	—
Potassium (mg/d)							
Baseline	42	2592 ± 170	42	2467 ± 160	0.394	0.790	0.763
Week 2	41	2615 ± 123	41	2366 ± 119	—	—	—
Week 12	41	2497 ± 105	41	2448 ± 150	—	—	—
Week 36	41	2528 ± 125	41	2557 ± 174	—	—	—
Sodium (mg/d)							
Baseline	42	3511 ± 209	42	3602 ± 262	<0.001	0.170	0.908
Week 2	41	3527 ± 205	41	2998 ± 141	—	—	—
Week 12	41	3126 ± 135	41	2790 ± 148	—	—	—
Week 36	41	3217 ± 159	41	3226 ± 181	—	—	—

[1] RGL, reduced glycemic load. Results for all variables except glycemic index and glycemic load are from analyses of 3-d food records; glycemic index and glycemic load results were derived from analyses of Willett food-frequency questionnaires.
[2] *P* values from repeated-measures ANOVA models with treatment, time, and treatment × time interaction as factors.
[3] *P* values from ANOVA models comparing treatments at each timepoint.
[4] $\bar{x}$ ± SEM (all such values).
[5] Calculated as the weighted average glycemic index value for all carbohydrate-containing foods in the diet.
[6] Calculated as each food's carbohydrate quantity (in g) multiplied by the respective glycemic index.

portion-controlled diet. No evidence was observed of any adverse effects on CVD risk markers.

Participants transitioned from the initial weight-loss period to a weight-maintenance phase by week 24. Subjects in both groups regained a portion of the maximum amount of weight lost: 1.9 and 1.8 kg in the RGL and control diets, respectively. At the end of the weight-maintenance period (week 36), differences between group losses of body weight and fat were no longer significant. In addition, no significant differences were observed in CVD risk markers, except for a larger increase in HDL cholesterol at week 36 in subjects in the RGL group than in the control group (3.8 and 1.9 mg/dL, respectively; $P = 0.037$). These findings suggest that, whereas the RGL diet produced greater initial losses of body weight and fat than did the portion-controlled diet, it showed no clear superiority with respect to its influence on CVD risk markers or maintenance of changes in body weight and composition.

The differential losses of body weight and fat during the weight-loss phase indicate that subjects in the RGL group experienced greater negative energy balance than did those in the control group. This difference must be explained by greater EE, lower energy intake, or some combination of these factors. The decline in resting EE during weight loss may be attenuated with an RGL diet (3, 5). Rats and mice fed an RGL diet, achieved by use of a low-GI chow (made with amylose), required more energy to maintain the same body weight than did animals fed high-GI chow (made with amylopectin) (27). Despite higher energy intake, animals fed the RGL diet had lower adiposity after 9–18 wk than did those fed the control diet (27). These findings support the view that an RGL diet may affect EE and partitioning in ways that would favor negative energy balance.

Dietary GL, insulin secretion, and substrate oxidation may interact to influence appetite. An RGL diet is likely to produce a lower average daylong insulin concentration (3, 28–30). This is especially true for persons with insulin resistance and compensatory hyperinsulinemia, both of which are characteristic features of obesity (31–33).

In persons with excess adiposity, the ability of insulin to promote glycogenesis is impaired to a greater extent than is its ability to promote glucose oxidation (31). In an insulin-resistant person, a high-GL diet would be expected to produce extended periods of hyperinsulinemia that would, in turn, increase whole-body carbohydrate oxidation and depress fat oxidation (34). Because it has been shown that increased hepatic fat oxidation is associated with lower hunger and food intake in animals and humans (7, 35–37), the reduced fat oxidation associated with hyperinsulinemia may lead to an increase in food intake.

Protein consumption tended to be higher in the RGL group than in the control group. Protein appears to have greater ability than does carbohydrate to promote satiety and reduce subsequent food intake (38–40). In addition, numerous studies have shown elevated thermic responses to meals and higher 24-h EE with increased protein intake (40). Diets with higher protein content have also been found to produce greater weight loss in some trials, which may be accounted for by these effects (40–42).

Previous studies showed that 20–30% of weight lost by dieting is typically fat-free mass (43). In this study, loss of fat-free mass was significantly larger in the RGL group at week 36 (≈51% and 41% of weight lost in the RGL and control groups, respectively), even after statistical adjustment for total weight lost ($P = 0.037$). The greater reduction in fat-free mass is of potential concern, because this reduction could result in attenuation of total daily EE if attributable to a greater loss of lean tissue (44). Because DXA cannot distinguish between changes in nonbone lean tissue and those in fluid, the degree to which fluid loss may have contributed to the loss of fat-free mass is unclear.

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TABLE 4
Cardiovascular disease risk markers by time point and treatment group[1]

Variable	RGL group		Control group		*P*		
					Time[2]	Treatment × time[2]	Pairwise[3]
	n		*n*				
Total cholesterol (mg/dL)							
Baseline[4]	42	199.3 ± 4.5[5]	42	206.5 ± 6.5	<0.001	0.153	0.362
Δ at week 12[6]	39	−12.2 ± 2.7	38	−8.3 ± 3.7	—	—	—
Δ at week 36[7]	39	−1.5 ± 3.9	38	−3.0 ± 2.9	—	—	—
LDL cholesterol (mg/dL)							
Baseline	42	117.6 ± 4.2	42	123.4 ± 5.7	<0.001	0.367	0.411
Δ at week 12[6]	39	−7.0 ± 2.2	38	−3.6 ± 2.9	—	—	—
Δ at week 36[7]	39	−2.8 ± 3.2	38	−1.9 ± 2.9	—	—	—
HDL cholesterol (mg/dL)							
Baseline	42	56.2 ± 2.0	42	56.4 ± 2.0	<0.001	0.013	0.982
Δ at week 12[6]	39	−0.2 ± 1.2	38	−2.1 ± 0.9	—	—	0.390
Δ at week 36[7]	39	3.8 ± 1.4	38	1.9 ± 0.8	—	—	0.037
Total:HDL cholesterol							
Baseline	42	3.7 ± 0.1	42	3.8 ± 0.2	<0.001	0.208	0.610
Δ at week 12[6]	39	−0.2 ± 0.1	38	0.0 ± 0.1	—	—	—
Δ at week 36[7]	39	−0.3 ± 0.1	38	−0.2 ± 0.1	—	—	—
Triacylglycerol (mg/dL)							
Baseline	42	127.1 ± 8.3	42	134.0 ± 10.6	0.003	0.139	0.936
Δ at week 12[6]	39	−24.8 ± 5.3	38	−11.5 ± 6.5	—	—	—
Δ at week 36[7]	39	−12.5 ± 5.2	38	−15.5 ± 8.9	—	—	—
Glucose (mg/dL)							
Baseline	43	95.3 ± 1.3	43	95.2 ± 1.7	0.035	0.237	0.791
Δ at week 12	39	−2.9 ± 1.3	39	−0.3 ± 1.1	—	—	—
Δ at week 36	39	−1.1 ± 1.7	39	2.6 ± 1.4	—	—	—
Insulin (mU/L)							
Baseline	43	10.4 ± 1.0	43	9.0 ± 1.2	0.003	0.502	0.061
Δ at week 12	39	−0.4 ± 1.1	39	0.9 ± 1.1	—	—	—
Δ at week 36	38	1.1 ± 1.0	39	2.4 ± 0.7	—	—	—
HOMA insulin resistance							
Baseline	42	2.4 ± 0.2	42	2.1 ± 0.3	0.003	0.534	0.057
Δ at week 12[6]	39	−0.1 ± 0.3	39	0.2 ± 0.3	—	—	—
Δ at week 36[7]	39	0.3 ± 0.3	39	0.7 ± 0.2	—	—	—
Systolic blood pressure (mm Hg)							
Baseline	43	112.7 ± 1.6	43	114.7 ± 1.6	0.372	0.367	0.376
Δ at week 12[6]	42	−0.6 ± 2.0	42	−1.2 ± 2.3	—	—	—
Δ at week 36[7]	42	0.2 ± 1.7	42	0.1 ± 2.2	—	—	—
Diastolic blood pressure (mm Hg)							
Baseline	43	74.4 ± 1.5	43	73.4 ± 1.1	0.016	0.455	0.615
Δ at week 12[6]	42	−3.3 ± 1.8	42	−0.6 ± 1.4	—	—	—
Δ at week 36[7]	42	−4.1 ± 1.8	42	−1.6 ± 1.3	—	—	—

[1] RGL, reduced glycemic load; HOMA, homeostasis model assessment.
[2] *P* values from repeated-measures ANOVA models with treatment, time, and treatment × time interaction as factors.
[3] *P* values from ANOVA models comparing treatments at each timepoint.
[4] Average of values obtained at weeks −1 and 0.
[5] $\bar{x}$ ± SEM (all such values).
[6] Average of values obtained at weeks 10 and 12.
[7] Average of values obtained at weeks 32 and 36.

Because insulin stimulates renal sodium and water reabsorption, an RGL diet may be expected to produce fluid loss (10, 45–47). However, insulin is also a growth factor that enhances protein synthesis and inhibits protein catabolism in muscle tissues (48, 49), and thus some greater loss of lean tissue may also have occurred if total circulating insulin exposure was reduced. Additional research will be needed to differentiate between changes in the masses of fluid and those in lean tissue with consumption of RGL diets.

A common concern expressed regarding carbohydrate-restricted diets is that they may be associated with higher consumption of saturated fatty acids and cholesterol, which could lead to higher LDL cholesterol. In a meta-analysis comparing low-carbohydrate with low-fat weight-loss diets, Nordmann et al (19) found that both HDL (4.6 mg/dL) and LDL (5.4 mg/dL) cholesterol were higher after 6 mo in subjects assigned to the low-carbohydrate diet. In the current study, reported saturated fat and cholesterol intakes were

higher in the RGL diet group than in the control group. Whereas the increase in HDL cholesterol at 36 wk was greater in the RGL diet group than in the control group (3.8 and 1.9 mg/dL, respectively; $P = 0.037$), a finding that is consistent with the results of the meta-analysis, no difference was found between the groups in the mean change in LDL cholesterol (-2.8 and -1.9 mg/dL, respectively; $P = 0.836$).

Jenkins et al (50) showed that, under conditions of identically matched food intake, a lower rate of carbohydrate absorption, elicited by consumption of 17 snacks as opposed to 3 larger meals, was associated with a 28% lower average insulin concentration and a mean reduction of 13.5% in LDL-cholesterol concentrations. Sloth et al (14) also found that ad libitum consumption of a low-GI diet resulted in a 10% decrease in LDL cholesterol, whereas consumption of a high-GI diet matched for macronutrient and fiber content per 1000 kcal resulted in a slight (2%) rise in LDL cholesterol.

Insulin has a stimulatory effect on VLDL synthesis (32). Therefore, a smaller integrated insulin response may reduce VLDL synthesis and entry into the circulation (5, 51). In the absence of changes in the rates of VLDL conversion to LDL or of LDL removal from the circulation (or both), a reduction in the concentration of LDL cholesterol would be expected. However, greater intakes of saturated fats and cholesterol can suppress hepatic LDL uptake (52), which may explain the absence of a larger reduction in LDL cholesterol in subjects consuming the RGL diet than in those consuming the control diet (53).

In summary, the results of the current trial showed that, in free-living, overweight and obese subjects, an ad libitum RGL diet produced greater losses of body weight and fat than did a traditional, portion-controlled diet during an initial weight-loss period. Weight regain from the point of maximal weight loss did not differ between treatments. However, the differences in body weight and body fat responses between groups were no longer significant at the end of the weight-maintenance phase of the trial (week 36). No evidence was present of any adverse effects of the RGL diet on CVD risk factors. Therefore, the results of the current study suggest that an ad libitum RGL diet is a reasonable alternative to a low-fat, portion-controlled weight-loss diet. Additional research is warranted to clarify the mechanisms responsible for the greater initial losses of body weight and fat associated with the RGL diet, to evaluate the persistence of those losses over longer treatment periods, and to obtain greater insight into strategies that would improve long-term weight-loss maintenance.

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Exhibit G

OBESITY

Dietary and Developmental Influences

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5 Conclusions

WHEN TO INTERVENE: CRITICAL PERIODS IN THE DEVELOPMENT OF OBESITY

The Intrauterine Environment

The risk of obesity is cumulative for each individual, and exposure begins at conception. Women who are obese, still growing, or diabetic provide an intrauterine environment for their fetus that differs from women who do not have these characteristics. The intrauterine environment appears to influence later risk for obesity through various mechanisms, often referred to as "fetal programming," which include changes in the central nervous system, in ratio of fat to lean body mass, in insulin metabolism, and in other hormonal factors. High birth weight (HBW) and exposure to diabetes *in utero* are both consistently associated with higher BMI later in life (attained BMI). However, when adjusted for confounders such as parental BMI, gestational age, and socioeconomic status, the relationship between birth weight and attained BMI is less robust, and recent studies suggest that the greater attained BMI may reflect greater lean body mass rather than fat mass. Low birth weight (LBW) is more consistently associated with attained BMI and has been implicated as a risk factor for central adiposity and risk of chronic disease. LBW followed by rapid "catch-up" growth in childhood appears to pose an additional risk for adult obesity. Furthermore, rates of LBW have risen while rates of HBW have fallen between 1980 and 2001. Taken as a whole, these finding suggest that the intrauterine environment plays an important, albeit complex, role in the etiology of obesity.

Infancy and Childhood

Growth patterns and feeding practices in infancy and early childhood add to the cumulative risk of obesity. A rapid rate of weight gain in infancy and early childhood (more commonly associated with formula than breastfeeding) may increase risk of obesity. Rate of weight gain in the first four months of life has been reported to be associated with overweight at age seven even after adjusting for birth weight and weight attained at one year, and children who gain weight for age faster than height for age in the first two years of life have higher BMI, body fat, and waist circumference at age five. The timing of adiposity rebound (the point in early childhood, usually between 4–8 years of age, when BMI starts to climb again after reaching a nadir) may be a particularly critical period. A trend toward earlier adiposity rebound has been reported over the last few decades, and children who experience an earlier adiposity rebound are at an increased risk for becoming overweight. It has been hypothesized that dietary and other lifestyle factors, including formula feeding, that stimulate rapid growth and early maturation may contribute to early adiposity rebound, but the extent to which the timing of adiposity rebound can be altered is unknown. Because overweight tends to track over time for children, an overweight child is more likely than a child of normal weight to be obese as an adult. This relationship gets stronger as children grow older. In this regard, all of childhood might be considered as a critical period for the development of adult weight status.

Puberty

Children who are growing more rapidly than their peers tend to mature earlier as well. Various studies suggest trends over the last several decades toward early onset of puberty and menarche

among girls of all racial groups studied. Observational studies consistently find an association between early puberty and higher adiposity. The relationship between puberty and adiposity is probably bidirectional. Early puberty may contribute to increased adiposity by nature of increased levels of hormones that stimulate the accumulation of body fat. Also, puberty is frequently associated with reduced levels of physical activity. Conversely, higher adiposity may stimulate hormones that trigger early maturation. It appears, therefore, that early influences set the stage for rapid growth and early maturation that tend to "snowball" as the child grows. Factors favoring increased adiposity at any stage, and the increase in adiposity itself, tend to predispose to continuing risk in subsequent stages of development.

Pregnancy and the Postpartum Period

Adult women experience particular life course factors that have been implicated as contributors to increased adiposity, namely pregnancy. Weight gained during pregnancy has increased during the last several decades. Although most women do not retain substantial weight due to pregnancies, a subset of women appears to be especially susceptible to postpartum weight retention. Women who gain more during pregnancy, lower-income women, adolescents who are still growing, and African-American women are at higher risk for weight retention with pregnancy. Despite the significant energy expenditure involved in producing breast milk, neither secular trends nor observational studies suggest a relationship between lactation and adiposity. Studies, however, are limited and confounded by many factors, including the probability that women who choose to formula feed are more likely to return to work, smoke cigarettes, and diet to lose weight.

Menopause

Weight gain during menopause is also highly variable but, on the whole, observational studies fairly consistently find an association between menopausal status and increased body fat, particularly abdominal fat. These associations are thought to be due to hormonal changes that favor the central deposition of body fat as well as an accelerated decline in resting metabolic rate. Accumulation of body fat can be exacerbated by declines in physical activity associated with menopause.

The Elderly

The rates of overweight experienced by older Americans appear to be due to the cumulative life time risk of becoming overweight rather than to a distinctly rapid rate of gain during the later years of life. Indeed, rates of overweight and obesity tend to level off during the seventh decade of life (60–69 years) and start to decline in the eighth decade (70+ years). It appears that weight history is a better predictor of disease than current weight among the elderly, and fat acquired late in life may not be as harmful as excess adiposity experienced over a lifetime. In fact, overweight may have a protective effect for the elderly, and the healthy weight range may therefore be somewhat higher than for other age groups. This should not be misinterpreted to suggest excess body weight is of no concern to the elderly. At a certain level, excess weight can impede physical activity, increase insulin resistance, and increase the risk for disability. Given that changes in body fat distribution, loss of muscle mass, and decreases in total energy expenditure are particular concerns for the elderly, a focus on maintaining a physically active lifestyle is particularly important for this age group.

IMPLICATIONS

The literature concerning the influences of life cycle on the development of obesity strongly point to the need for early prevention efforts. To be effective at *preventing* obesity, interventions should begin with preconception and continue through pregnancy to decrease the likelihood of fetal obesity programming. Early childhood is equally critical for preventing rapid growth and metabolic sequelae that increase the risk of higher adiposity, as well as early puberty, which, in turn, creates additional risk for excess weight gain. Tracking of overweight through childhood and into adulthood provides further evidence that *prevention* must begin early. With cumulative exposure through childhood, whereby one insult then increases the risk for the next insult and so on, the course may be hard to reverse by the time a child reaches puberty. Therefore, prevention efforts must not only begin preconception but must continue throughout childhood.

The biggest increases in rates of overweight and obesity among adults occur during early adulthood and tend to level off as the population ages. Therefore *prevention* efforts would probably have the biggest impact among younger adults. Pregnancy may be the most important discrete life cycle factor during adulthood, especially for women who are susceptible to excessive weight gain during pregnancy. The effects of the mother's weight gain and health on the health of the fetus provide additional rationale for a focus on pregnancy and the postpartum period. It is not yet established if lactation provides protection against overweight for the mother; however, other benefits to the mother and child are indisputable. Menopause poses an additional risk for women. Maintaining an active lifestyle is particularly important for menopausal women, because women at this stage of life are at increased risk for increases in abdominal fat mass, decreases in lean body mass, and decreases in resting metabolic rate, all of which can be improved with increased physical activity. The elderly tend to have the lowest risk for becoming obese, and the healthy weight range among the elderly may be higher than for younger age groups. As with menopausal women, a focus on physical activity is particularly important for this age group; prevention of weight gain is probably less of a concern. Weight loss may be beneficial for older adults whose weight is associated with comorbidities. However, the merit of weight loss for overweight elderly was not addressed by this review.

WHAT TO RECOMMEND: DIETARY INFLUENCES ON ENERGY BALANCE

This review examined the evidence relating 28 different dietary factors to adiposity with the intent of determining the dietary composition and eating behaviors that would be most conducive to the prevention of overweight. These dietary factors are listed in Tables 5.1 and 5.2, which summarize the consistency of evidence from the various types of studies examined (see Chapter 3). Eleven of these factors (Table 5.1) were found to have moderately consistent evidence supporting a relationship with adiposity. These include

1. dietary fat
2. total carbohydrate
3. dietary fiber
4. fruits and vegetables
5. sweetened beverages
6. calcium
7. dairy
8. dietary patterns
9. restaurant-prepared/fast foods
10. breakfast skipping
11. breastfeeding

TABLE 5.1
Summary of the Nature and Consistency of Evidence Supporting the Relationship between Various Dietary Factors and Lower Adiposity: Factors for Which Support Was Moderate

Nutrient/ Target Behavior[1] (Total number of observational studies/prevention trials)	Secular Trends Support Relationship?	Mechanisms Support Relationship?	Observational Studies Support Relationship?			Prevention Trials Support Relationship?		Conclusion: Consistency of Evidence Supporting Relationship
			Longitudinal	U.S. Nationally Representative Cross-Sectional	Other Cross-Sectional or Case-Control	Randomized Controlled Trials	Other	
↓ Dietary fat	No	Yes						Moderate
28/10					*Adults*			
Adults			Inconclusive	Inconclusive	Yes	Yes	No studies	
47/14								
Children					*Children*			
			Inconclusive	Inconclusive	Inconclusive	Inconclusive	Inconclusive	
↑ Total% carbohydrate	No	Yes						Moderate
20/6					*Adults*			
Adults			Inconclusive	Yes	Yes	Yes	No studies	
32/3								
Children					*Children*			
			Inconclusive	No studies	Inconclusive	Inconclusive	No studies	
↑ Dietary fiber	No	Yes						Moderate
8/5					*Adults*			
Adults			Yes	No studies	Yes	Yes	No studies	
7/5								
Children					*Children*			
			Inconclusive	No studies	Yes	Inconclusive	Inconclusive	
↑ Calcium intake	No — adults	Yes						Moderate
7/14[2]	Yes — children				*Adults*			
Adults			Yes	Yes	Inconclusive	Inconclusive	Yes	
4/7[2]								
Children					*Children*			
			Yes	No studies	Yes	Inconclusive	No studies	
↑ Dairy intake	No — total dairy	Yes						Moderate
11/7	Yes— milk				*Adults*			
Adults			Yes	Yes	Inconclusive	Inconclusive	Yes	
5/3								
Children					*Children*			
			No studies	Yes	Yes	Inconclusive	No studies	

↑ Vegetable and fruit intake 22/3 Adults 10/5 Children	No	Yes	***Adults***				Moderate
			Inconclusive	Yes	Yes	Inconclusive	Inconclusive
			Children				
			No studies	Yes	Inconclusive	Inconclusive	Inconclusive
↓ Sweetened beverage intake 1/0 Adults 9/2 Children	Yes	Yes	***Adults***				Moderate
			Inconclusive	No studies	No studies	No studies	No studies
			Children				
			Yes	Yes	Inconclusive	Inconclusive	Inconclusive
↓ Eating restaurant-prepared food 9/0 Adults 4/0 Children	Yes	Yes	***Adults***				Moderate
			Yes	Yes	Yes	No studies	No studies
			Children				
			No studies	No studies	Inconclusive	No studies	No studies
Δ Dietary patterns 23/* Adults 0/* Children	Yes	Yes	***Adults***				Moderate
			Yes	Inconclusive	Yes	Yes[1]	Yes[3]
			Children				
			No studies	No studies	No studies	Inconclusive[3]	Inconclusive[3]
↓ Breakfast skipping 7/0 Adults 9/0 Children	Yes	Inconclusive	***Adults***				Moderate
			No studies	Yes	Yes	No studies	No studies
			Children				
			No studies	Yes	Yes	No studies	No studies
↑ Breastfeeding 3/0 Adults 28/0 Children	No	Yes	***Adults***				Moderate
			Inconclusive	No studies	Inconclusive	No studies	No studies
			Children				
			Yes	Inconclusive	Yes	No studies	No studies

Notes:

[1]Description of criteria used for summary table is located in the methods section.

[2]The five studies included in the meta-analysis by Davies (2000) are included individually in the above count by study type.

[3]Based on trials reviewed in other sections of this document.

TABLE 5.2
Summary of the Nature and Consistency of Evidence Supporting the Relationship between Various Dietary Factors and Lower Adiposity: Factors for Which Evidence Was Inconclusive

Nutrient/ Target Behavior[1] (Total number of observational studies/prevention trials)	Secular Trends Support Relationship?	Mechanisms Support Relationship?	Observational Studies Support Relationship?			Prevention Trials Support Relationship?		Conclusion: Consistency of Evidence Supporting Relationship
			Longitudinal	U.S. Nationally Representative Cross-Sectional	Other Cross-Sectional or Case-Control	Randomized Controlled Trials	Other	
↓ Energy density	Inconclusive	Yes						Inconclusive
4/0					*Adults*			
Adults			No studies	Inconclusive	Yes	No studies	No studies	
0								
Children					*Children*			
			No studies	No studies	No studies	No studies	No studies	
↑ Complex CHO	No	Yes						Inconclusive
4/1					*Adults*			
Adults			No studies	No studies	Yes	Inconclusive	No studies	
0/0								
Children					*Children*			
			No studies	No studies	No studies	No studies	No studies	
↓ Total sugar/simple CHO	Yes	Yes						Inconclusive
8/1					*Adults*			
Adult			No studies	No studies	No	Inconclusive	No studies	
3/0								
Children					*Children*			
			No studies	No studies	Inconclusive	No studies	No studies	
↓ Added sugar/sucrose	Yes	Yes						Inconclusive
5/0					*Adults*			
Adult			Inconclusive	Inconclusive	Inconclusive	No studies	No studies	
2/3								
Children					*Children*			
			No studies	Inconclusive	No studies	Inconclusive	Inconclusive	
↓ Intrinsic sugar	None	Yes						Inconclusive
1/0					*Adults*			
Adults			No studies	No studies	Inconclusive	No studies	No studies	
0/0								
Children					*Children*			
			No studies	No studies	No studies	No studies	No studies	

↓ Dietary protein 12/1 Adults 30/2 Children	No	Inconclusive						Inconclusive
					Adults			
			Inconclusive	No studies	Yes	Inconclusive	No studies	
					Children			
			Inconclusive	No studies	Inconclusive	Inconclusive	No studies	
↑ Iron or zinc intake 2/0 Adults 1/0 Children	Inconclusive	Inconclusive						Inconclusive
					Adults			
			Inconclusive	No studies	Inconclusive	No studies	No studies	
					Children			
			No studies	No studies	Inconclusive	No studies	No studies	
↑ Antioxidant vitamin intake 4/0 Intake 16/0 Blood	No	No						Inconclusive
					Intake			
			Inconclusive	No studies	Inconclusive	No studies	No studies	
					Blood			
			No studies	Yes[2]	Yes[2]	No studies	No studies	
↓ 100% fruit juice intake 0/0 Adult 9/0 Children	No	Yes						Inconclusive
					Adults			
			No studies	No studies	No studies	No studies	No studies	
					Children			
			No	No	Yes	No studies	No studies	
↓ Variety of food intake 2/1 Adults 3/0 Children	Yes	Inconclusive						Inconclusive
					Adults			
			No studies	No studies	Inconclusive	Inconclusive	No studies	
					Children			
			No studies	Inconclusive	Inconclusive	No studies	No studies	
↓ Reduced fat food product intake 3/9 Adults 2/0 Children	Yes	No						Inconclusive
					Adults			
			No studies	Inconclusive	Inconclusive	No	No	
					Children			
			Inconclusive	No studies	Inconclusive	No studies	No studies	

Exhibit H

REVIEW

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Childhood obesity: behavioral aberration or biochemical drive? Reinterpreting the First Law of Thermodynamics

Robert H Lustig

SUMMARY

Childhood obesity has become epidemic over the past 30 years. The First Law of Thermodynamics is routinely interpreted to imply that weight gain is secondary to increased caloric intake and/or decreased energy expenditure, two behaviors that have been documented during this interval; nonetheless, lifestyle interventions are notoriously ineffective at promoting weight loss. Obesity is characterized by hyperinsulinemia. Although hyperinsulinemia is usually thought to be secondary to obesity, it can instead be primary, due to autonomic dysfunction. Obesity is also a state of leptin resistance, in which defective leptin signal transduction promotes excess energy intake, to maintain normal energy expenditure. Insulin and leptin share a common central signaling pathway, and it seems that insulin functions as an endogenous leptin antagonist. Suppressing insulin ameliorates leptin resistance, with ensuing reduction of caloric intake, increased spontaneous activity, and improved quality of life. Hyperinsulinemia also interferes with dopamine clearance in the ventral tegmental area and nucleus accumbens, promoting increased food reward. Accordingly, the First Law of Thermodynamics can be reinterpreted, such that the behaviors of increased caloric intake and decreased energy expenditure are secondary to obligate weight gain. This weight gain is driven by the hyperinsulinemic state, through three mechanisms: energy partitioning into adipose tissue; interference with leptin signal transduction; and interference with extinction of the hedonic response to food.

KEYWORDS addiction, insulin, leptin resistance, obesity, starvation

REVIEW CRITERIA

I searched for original articles focusing on energy balance and obesity in MEDLINE and PubMed from 1994 to 2005. The search terms I used were "insulin", "insulin resistance", "leptin", and "leptin resistance". All papers identified were English-language full-text papers. I also searched the reference lists of identified articles for further papers.

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INTRODUCTION

We are in the midst of an unprecedented rise in prevalence and severity of obesity, especially in children.[1] Although common wisdom dictates that obesity is an interaction between genetics and environment, the gene pool has not changed in the last 30 years, but the environment has. Examination of the BMI distribution histogram[2] demonstrates that even those at the lower end of the BMI curve are gaining weight. Whatever is happening is happening to everyone, suggesting an environmental trigger. This observation is also reflected in the increased prevalence of obesity in developing countries.[3] This review will address the relationship between changes in the environment and the neuroendocrinology of human energy balance, particularly as it applies to the current childhood obesity epidemic.

THE FIRST LAW OF THERMODYNAMICS

Energy balance obeys the First Law of Thermodynamics, which states: "The energy within a closed system remains constant". This law is usually interpreted as "If you eat it (energy intake), you must burn it (energy expenditure), or you will store it (weight gain)". This view is buttressed by studies of increased caloric intake[4] and decreased energy expenditure[5] in children. This interpretation of the First Law of Thermodynamics promulgates the idea that obesity is the result of pathologic behavior on the part of the obese person, which invokes the concept of personal responsibility, and permits absolution of responsibility by governments and the business community. The concept of personal responsibility is, however, not tenable in children. No child chooses to be obese. Obese children are ostracized by their peers, and their quality of life, as measured by self-reported distress, is comparable to those receiving cancer chemotherapy.[6] Young children, among whom obesity is rampant,[1] are not responsible for their food choices and are incapable of accepting personal responsibility.

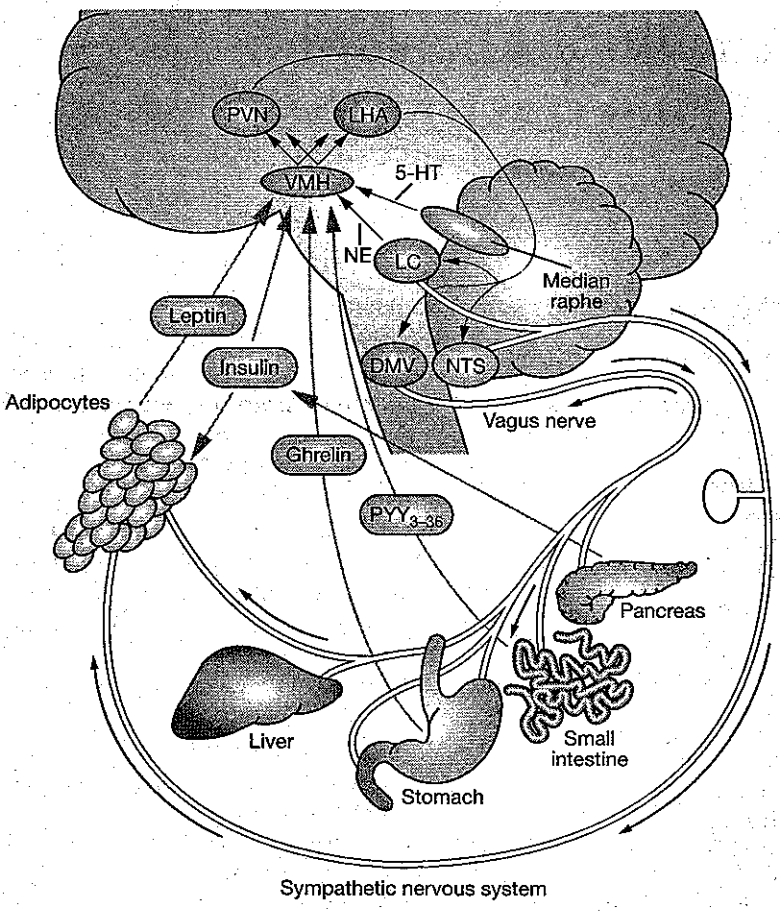

Figure 1 The homeostatic pathway of energy balance. Afferent (blue), central (brown), and efferent (white) pathways are delineated. The hormones insulin, leptin, ghrelin, and peptide YY_{3-36} provide afferent information to the ventromedial hypothalamus, relating to short-term energy metabolism and energy sufficiency. From there, the ventromedial hypothalamus elicits anorexigenic (α-melanocyte stimulating hormone, cocaine-amphetamine regulated transcript) and orexigenic (neuropeptide Y, agouti-related protein) signals to the melanocortin 4 receptor in the paraventricular nucleus and lateral hypothalamic area. These signals lead to efferent output via the locus coeruleus and the nucleus tractus solitarius, which activates the sympathetic nervous system and causes adipocytes to undergo lipolysis, or via the dorsal motor nucleus of the vagus, which activates the vagus nerve and causes energy storage, both by increasing pancreatic insulin secretion, and (in rodents) by increasing adipose-tissue sensitivity to insulin.[9] Reproduced with permission from reference 9 © (2001) Elsevier Inc. Abbreviations: 5-HT, serotonin (5-hydroxytryptamine); DMV, dorsal motor nucleus of the vagus; LC, locus coeruleus; LHA, lateral hypothalamic area; NE, norepinephrine; NTS, nucleus tractus solitarius; PVN, paraventricular nucleus; PYY_{3-36}, peptide YY_{3-36}; VMH, ventromedial hypothalamus.

BEHAVIOR IS REALLY BIOCHEMISTRY

If obesity is a result of behavior, then behavior and/or lifestyle modification (BLM) should be effective in mitigating the process. Indeed, as a result of some individual successes,[7] BLM has become the cornerstone of therapy; however, BLM is not successful in the majority of obese children.[8] Behavior is defined as "a stereotyped motor response to a physiological stimulus". This definition implies that there are physiological bases underpinning behavior. This relationship is evident in many medical disorders, such as diabetes insipidus, narcolepsy, and schizophrenia. So, what is the physiology behind gluttony and sloth? And how does our current environment bring about these conditions?

THE HOMEOSTATIC PATHWAY OF ENERGY BALANCE

The hypothalamus orchestrates the neuroendocrine control of energy balance, via a complex homeostatic pathway that comprises three arms[9] (Figure 1). The first arm consists of afferent signals to the central nervous system (CNS). The ventromedial hypothalamus (VMH), consisting of the arcuate and ventromedial nuclei, contains first-order neurons with hormone receptors that receive peripheral signals related to adiposity (leptin), metabolism (insulin), hunger (ghrelin), and satiety (peptide YY_{3-36}). The second arm consists of second-order neurons, which transduce these hormonal signals to the paraventricular nucleus (PVN) and lateral hypothalamic area. These signals are either anorexigenic (e.g. α-melanocyte stimulating hormone, cocaine-amphetamine-regulated transcript) or orexigenic (e.g. neuropeptide Y, agouti-related protein). The PVN and lateral hypothalamic area integrate these signals via melanocortin 4 receptor (MC4-R) occupancy to alter caloric intake and energy expenditure.[10] The third arm consists of efferent signals via the autonomic nervous system. The sympathetic nervous system (SNS) promotes energy expenditure, whereas the vagus nerve promotes energy storage[11] (see below). Insulin is part of both the afferent and efferent pathways. Unraveling its dual role provides valuable insights into the pathogenesis of obesity.[12]

The afferent pathway

Leptin as an afferent signal

The adipocyte hormone leptin conveys a signal of peripheral energy sufficiency to neurons in the arcuate nucleus expressing the leptin receptor.[13] Leptin increases α-melanocyte stimulating hormone and inhibits neuropeptide Y, which reduces food intake and increases SNS activity.[14] Leptin is a permissive, afferent signal for the initiation of high-energy processes, such as puberty and pregnancy.[13]

Insulin as an afferent signal, and its similarity to leptin

Insulin receptors co-localize to the same subpopulation of arcuate neurons as do leptin receptors.[15] Insulin gains access to the CNS via a saturable transporter.[16] In animals, acute intracerebroventricular insulin infusion decreases feeding behavior, induces satiety, and activates the SNS, similarly to leptin.[17] Insulin and leptin both acutely activate the insulin receptor substrate 2–phosphatidylinositol 3 kinase (IRS-2–PI3K) second-messenger system in VMH neurons,[18] which increases neurotransmission of the central anorexigenic signaling pathway. The importance of insulin action in the CNS was demonstrated by production of mice in which the insulin receptor was knocked out specifically in the brain; these mice cannot transduce CNS insulin signals,[19] and they become hyperphagic, obese, and infertile—similar to leptin-deficient (*ob/ob*) and leptin-resistant (*db/db*) mice. Similarly, knockout of IRS-2 within the CNS interferes with the afferent signal and produces obesity.[20,21] These data suggest that insulin mediates an acute afferent signal similar to that mediated by leptin.[22]

The efferent pathway

The sympathetic nervous system and energy expenditure

Anorexigenic pressure by leptin, which alters MC4-R occupancy in the PVN,[10] leads to decreased food intake, whereas MC4-R activation in other CNS sites promotes increased energy expenditure, through activation of the SNS[23] (Figure 2A). For instance, leptin administration to *ob/ob* mice promotes increased lipolysis in brown adipose tissue, increased thermogenesis, and increased movement, all associated with increased energy expenditure and weight loss;[24] however, this effect is not seen in the Zucker rat strain, which has a mutation in the leptin receptor.[25]

SNS activation increases energy expenditure through glycogenolysis, and through glucose and fatty acid oxidation via β_2-adrenergic receptor activation in skeletal muscle.[26] SNS activation also promotes lipolysis in adipocytes, through activation of the β_3-adrenergic receptor, (which increases expression and activity of uncoupling proteins UCP 1 and UCP 2[23]) and via hormone-sensitive lipase, which is responsible for breakdown of triglyceride to free fatty acids (Figure 2B). Lastly, the SNS activates the α_2-adrenergic receptor on β cells and reduces insulin release (Figure 2C).

The vagus nerve and energy storage

Vagus nerve activation opposes SNS activation, and promotes energy storage. The vagus nerve slows the heart rate to reduce myocardial oxygen consumption, and it promotes peristalsis and energy substrate absorption from the alimentary tract. In rats, vagal efferent fibers have synapses on adipocytes (Figure 2B) and vagal activation improves insulin sensitivity and increase the clearance of energy substrate into adipose tissue (not yet demonstrated in humans).[27] Vagal modulation of β-cell function promotes insulin hypersecretion and increases substrate partitioning into adipose tissue (Figure 2C).[28] This action is exemplified by the M3 muscarinic receptor knockout mouse strain, which remains lean with low insulin secretion.[29]

The leptin 'set-point' and the starvation response

Examination of weight-loss patterns in response to BLM, and the effects of the obesity drugs dexfenfluramine, sibutramine, and orlistat demonstrate remarkable similarities.[30] Each exhibits a weight-loss phase of 4 months, followed by a plateau. Although reduced dietary intake and/or absorption continues, the plateau is inviolate. Although originally viewed as a result of noncompliance or drug tachyphylaxis, this plateau is actually due to a decline in resting energy expenditure (REE) that occurs in response to the decline in serum leptin, which offsets the reduced caloric intake. This reduction in REE is termed the "starvation response". Leibel *et al.*[31] showed that REE is dependent on nutritional status; in the energy-replete state, REE is 50 kcal/kg fat-free mass, whereas when weight is reduced, REE is reduced to 40–42 kcal/kg fat-free mass.[32] Thus, starvation results in a 20% increased efficiency of energy use.[33] Reductions in REE (e.g. that result from starvation or hypothyroidism) are associated with fatigue, malaise, decreased physical activity, and decreased quality of life. Conversely, factors that increase REE—primarily stimulants (e.g. caffeine, a phosphodiesterase inhibitor that stimulates fat oxidation)—increase wellbeing and physical activity.[34,35]

It has been postulated that each human has an individual leptin 'set-point'—a leptin concentration at which the hypothalamus perceives a state of energy sufficiency[13]—that is probably genetically determined. Leptin concentrations drop precipitously during periods of short-term

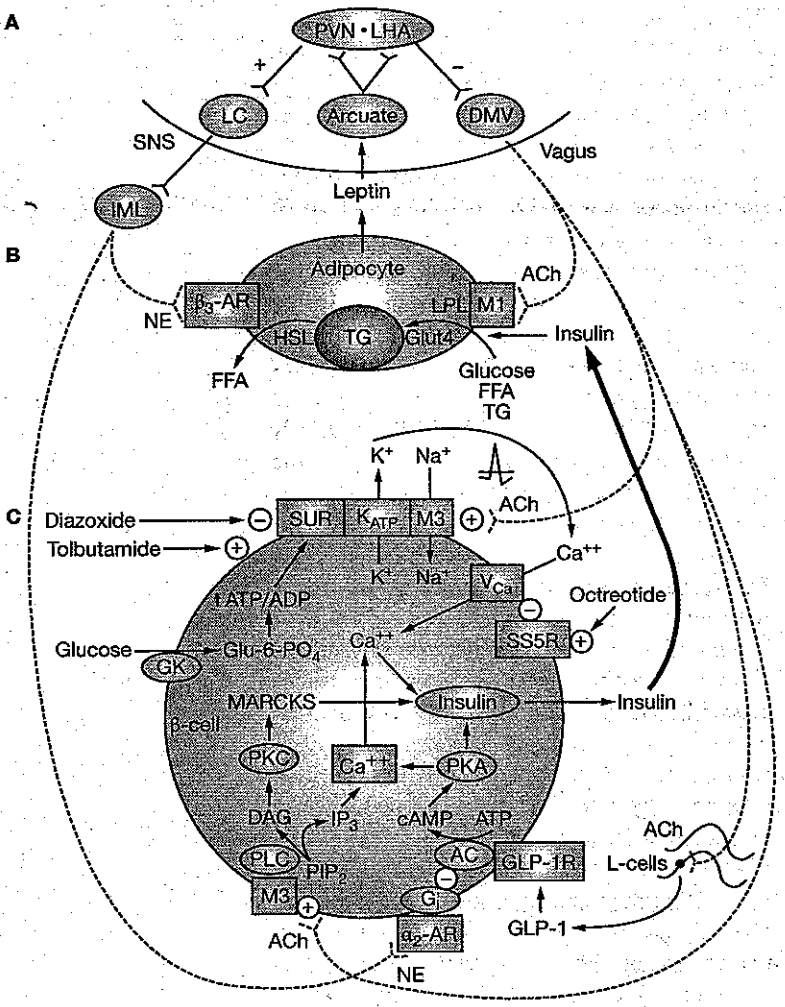

Figure 2 Legend opposite.

fasting (within 12 h), and decline faster than body fat stores are depleted.[36] This drop in leptin is interpreted by the hypothalamus as diminished energy reserve, which invokes the starvation response (Figure 2) and brings about alteration of efferent autonomic output in order to conserve energy.[37] Reduction in adrenergic tone reduces skeletal muscle energy usage and physical activity. Increased vagal tone reduces myocardial oxygen consumption, increases gastric motility (which increases appetite and alimentary absorption), increases adipose tissue insulin sensitivity, and increases pancreatic insulin secretion. These adaptations partition more energy to adipose tissue and raise leptin levels back to the energy-replete state.[11]

The concepts of the leptin set-point and the starvation response are exemplified by the leptin-deficient state. Both *ob/ob* mice and leptin-deficient humans exhibit a chronic starvation response, characterized by decreased SNS tone leading to decreased REE, decreased physical activity, and decreased reproductive capacity; along with increased vagal tone, which promotes increased insulin secretion and further weight gain.[38] Similarly, in the Zucker rat strain, increased vagal tone results in insulin hypersecretion that promotes weight gain.[39] Exogenous leptin administration above the leptin set-point re-establishes normal autonomic function, allowing for normal REE, increased physical activity, and weight loss due to lipolysis.

TWO FORMS OF CENTRAL NERVOUS SYSTEM LEPTIN RESISTANCE

Idiopathic obesity: 'functional' leptin resistance

This hypothetical CNS leptin set-point is dysfunctional in obesity; leptin levels are elevated, but do not trigger increased REE, lipolysis, or decreased food intake.[40] In the obese state, a higher level of leptin is required to signal the hypothalamus to maintain a normal REE. If energy intake declines, as occurs with dieting, leptin levels decline, triggering the starvation response; REE decreases, preventing further weight loss, with increased appetite, vagal activation, insulin hypersecretion, and increased energy storage. Exogenous leptin administration to obese individuals has only minor effects on weight loss because of CNS leptin resistance;[41] thus obesity is characterized as a functionally leptin-resistant state.[13]

Hypothalamic obesity: 'organic' leptin resistance

This phenomenon of CNS leptin resistance is recapitulated in the syndrome of hypothalamic obesity. Hypothalamic damage can be a sequel of cranial insult due to head trauma, posterior fossa brain tumors, surgery, or radiation.[42] A direct relationship between hypothalamic damage and BMI increase has been noted in survivors of childhood brain tumors.[43] Death of VMH neurons prevents normal leptin signal transduction, resulting in organic leptin resistance, which manifests as the starvation response. Decreased SNS tone[44] leads to decreased physical activity[45] and decreased energy expenditure. Conversely, increased vagal tone leads to increased pancreatic insulin

Figure 2 Central regulation of leptin signaling, autonomic innervation of adipocytes and β-cells, and the starvation response. **(A)** The arcuate nucleus transduces the peripheral leptin signal as one of sufficiency or deficiency. In leptin sufficiency, efferent fibers from the hypothalamus have synapses in the locus coeruleus, which stimulate the sympathetic nervous system. In leptin deficiency, efferent fibers from the hypothalamus stimulate the dorsal motor nucleus of the vagus nerve. **(B)** Autonomic innervation and hormonal stimulation of white adipose tissue. In leptin sufficiency, norepinephrine binds to the β_3-adrenergic receptor, which stimulates hormone-sensitive lipase, promoting lipolysis of stored triglyceride into free fatty acids. In leptin deficiency, vagus nerve mediated acetylcholine release activates the M1 receptor and increases adipose tissue insulin sensitivity (documented only in rats to date), promotes lipogenesis via uptake of glucose and free fatty acids, and promotes triglyceride uptake through activation of lipoprotein lipase. **(C)** Autonomic innervation and hormonal stimulation of β-cells. Glucose entering the cell is converted to glucose-6-phosphate by the enzyme glucokinase, generating ATP, which closes an ATP-dependent potassium channel and results in cell depolarization. A voltage-gated calcium channel opens, allowing intracellular calcium influx, which activates neurosecretory mechanisms leading to insulin vesicle exocytosis. In leptin sufficiency, norepinephrine binds to α_2-adrenoceptors on the β-cell membrane and stimulates inhibitory G proteins. Adenyl cyclase activity (and thus generation of its product, cyclic AMP) falls, which reduces protein kinase A levels and reduces insulin release. In leptin deficiency, vagus activation stimulates insulin secretion through three mechanisms.[28] First, acetylcholine binds to a M3 muscarinic receptor, opening a sodium channel, which augments the ATP-dependent cell depolarization, increases the calcium influx, and results in insulin vesicle exocytosis. Second, acetylcholine activates a pathway that increases protein kinase C levels, which also promotes insulin secretion. Third, vagus nerve fibers innervate L-cells of the small intestine, which secrete glucagon-like peptide 1, which activates protein kinase A and contributes to insulin-vesicle exocytosis. Octreotide binds to a somatostatin receptor on the β-cell that is coupled to the voltage-gated calcium channel. Octreotide, therefore, limits calcium influx and restricts the amount of insulin released in response to glucose.[11] Reproduced with permission from reference 28 © (2003) Springer Science and Business media. Abbreviations: + in circle and – in circle, positive and negative; α_2-AR, α_2-adrenergic receptor; β_3-AR, β_3-adrenergic receptor; AC, adenyl cyclase; ACh, acetylcholine; cAMP, cyclic AMP; Ca^{++}, calcium ions; DAG, diacylglycerol; DMV, dorsal motor nucleus of the vagus nerve; FFA, free fatty acids; G_i, inhibitory G protein; GK, glucokinase; GLP-1, glucagon-like peptide 1; GLP-1R, GLP-1 receptor; Glu-6-PO_4, glucose-6-phosphate; Glut4, glucose transporter 4; HSL, hormone-sensitive lipase; IML, intermediolateral cell column; IP_3, inositol triphosphate; K^+, potassium ions; K_{ATP} ATP-dependent potassium channel; LC, locus coeruleus; LHA, lateral hypothalamic area; LPL, lipoprotein lipase; M1 and M3, muscarinic receptors; MARCKS, myristoylated alanine-rich protein kinase C substrate; Na^+, sodium ions; NE, norepinephrine; PIP_2, phosphatidylinositol pyrophosphate; PKA, protein kinase A; PKC, protein kinase C; PLC, phospholipase C; PVN, paraventricular nucleus; SNS, sympathetic nervous system; SS5R, somatostatin receptor type 5; SUR, sufonylurea receptor; TG, triglyceride; V_{Ca}, voltage-gated calcium channel; VMH, ventromedial hypothalamus.

secretion[28] and increased adipocyte insulin sensitivity,[27] which promotes energy storage in adipose tissue. Patients manifest an extremely poor quality of life, with minimal physical activity; indeed parents complain that these are the most morbid sequelae such children experience.[42,45] Hypothalamic obesity is classically unresponsive to diet, exercise, and most pharmacologic manipulations.

PARADIGMS FOR IMPROVEMENT OF LEPTIN SENSITIVITY

If obesity is a state of CNS leptin resistance, then improving leptin sensitivity should result in weight loss and improved quality of life. Two strategies have so far been shown to improve leptin sensitivity in humans.

Forced weight loss

Rosenbaum *et al.*[46] employed inpatient energy restriction to generate 10% weight loss and induce the starvation response. In these individuals, leptin declined, and REE decreased, with commensurate decrease in serum T_3 levels. Exogenous administration of leptin in physiologic dosing to approximate the prestarvation leptin level resulted in further weight and fat decrease, along with return of REE and T_3 levels to the prestarvation state. In the prestarvation state, these individuals were resistant to physiologic concentrations of endogenous leptin, whereas in the weight-reduced state, they were responsive to the same concentrations of exogenously administered leptin—forced weight loss improved their inherent leptin sensitivity.

Insulin suppression

Hypothalamic obesity

Bray and Gallagher[47] posited that the weight gain in hypothalamic obesity was a result of the increased vagal tone after the VMH lesion, resulting in insulin hypersecretion that was evident on oral glucose tolerance testing (OGTT). My group hypothesized that suppression of β-cell insulin release should promote weight loss, despite the presence of 'organic' leptin resistance. We examined the effects of the somatostatin analog octreotide (an agonist of the somatostatin receptor type 5, which is present on β-cells and inhibits the voltage-gated calcium channel) (Figure 2C) in children with hypothalamic obesity. A pilot, open-label trial of subcutaneous octreotide 15 μg/kg per day for 6 months in eight patients[48] demonstrated a BMI loss commensurate with the degree of insulin suppression, along with decreased caloric intake, and subjective improvements in spontaneous physical activity and quality of life. A double-blind, placebo-controlled trial of octreotide in 20 individuals[49] resulted in insulin suppression and stabilization of BMI, decreased leptin levels, decreased caloric intake, increased spontaneous physical activity, and improvements in quality of life commensurate with the degree of insulin suppression. In other words, insulin suppression reversed the starvation response.

Adult obesity

In response to the success of octreotide in treating hypothalamic obesity, we then postulated that a subset of obese adults without CNS insult might also exhibit increased vagal tone and insulin hypersecretion, and might respond favorably to octreotide-LAR (long-acting release).[50] Treatment of 44 obese (mean BMI 44.1 kg/m^2) adults with octreotide-LAR (40 mg intramuscularly) at monthly intervals for 6 months resulted in significant weight (mean 12.6 kg) and BMI (mean 4.3 kg/m^2) loss in 8 individuals (the responders). These individuals manifested increased vagal tone, which correlated with insulin hypersecretion during the OGTT, as opposed to the 36 nonresponders who demonstrated insulin resistance. Indeed, octreotide-LAR suppressed insulin secretion only in the 8 responders.

Recalled caloric intake demonstrated an altered macronutrient preference, as responders selectively reduced their carbohydrate intake. In the responders, leptin concentration was reduced by 50%, but REE remained the same; so, when corrected for fat-free mass, REE increased. We used the change in REE:leptin ratio as a surrogate index of the change in leptin sensitivity in these individuals. Indeed, the REE:leptin ratio increased in those who lost weight. Linear regression analysis that compared the change in REE:leptin with the change in insulin response to OGTT demonstrated a significant negative correlation.[51] In other words, insulin suppression improved leptin sensitivity, as measured by this surrogate index. Notably, continued treatment of the responders for 1 year resulted in continued weight loss (mean 15.8 kg), with no ill effects. The beneficial effects of insulin suppression with octreotide-LAR, namely weight loss and improved quality of life, have been corroborated in a randomized, controlled trial of this agent in obese adults with insulin hypersecretion.[52]

Similarities between these paradigms

Forced weight loss improved leptin sensitivity, as measured by improvements in REE and thyroid hormone levels, in response to exogenous leptin. Insulin suppression using octreotide also improved leptin sensitivity, as measured by declining leptin concentrations with increased REE, allowing for weight loss and improved quality of life. Both strategies share at their core a reduction in systemic insulin concentrations. The similarity of outcome suggests that hyperinsulinemia may be a proximate cause of leptin resistance.

Molecular mechanisms of leptin resistance

The mechanism(s) of leptin resistance remain obscure. In rodents, diet-induced obesity (DIO) promotes defective leptin signal transduction, whether the leptin is administered peripherally or centrally;[53] however, DIO is also known to induce hyperinsulinemia.[54] Mouse models of improved leptin sensitivity with protection against weight gain have been demonstrated by knockout of the protein SOCS-3 (suppressor of cytokine signaling 3),[55] which inactivates the leptin receptor by dephosphorylating tyrosine 1138 (Figure 3).[56] Parenthetically, insulin induces SOCS-3,[57] which—as detailed below—could account for both insulin resistance and leptin resistance.

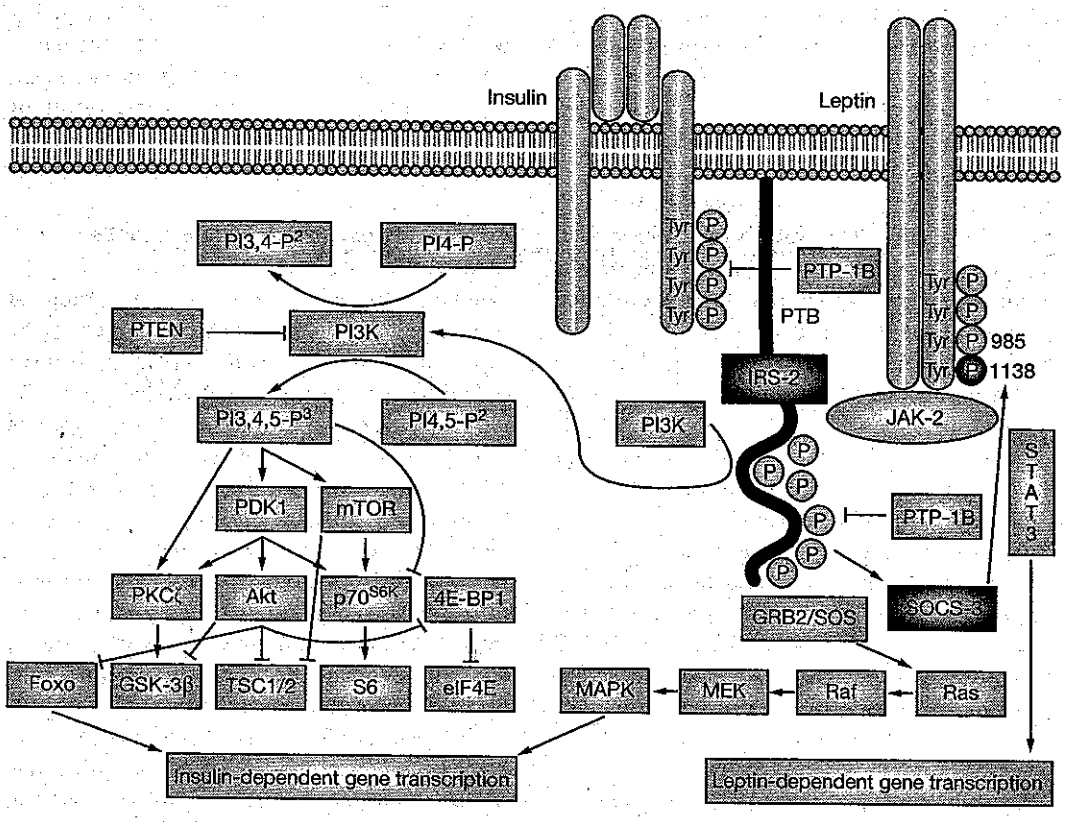

Figure 3 Overlap (depicted in black) between insulin and leptin signaling pathways in the ventromedial hypothalamic neuron. Insulin stimulates the insulin receptor substrate 2–phosphatidylinositol 3 kinase pathway, whereas leptin stimulates the Janus kinase 2–signal transduction and transcription 3 pathway; however, both the insulin receptor and the leptin receptor recruit the low-abundance-message second messenger insulin receptor substrate 2. Lack of available insulin receptor substrate 2 for the leptin receptor following hyperinsulinemia could result in defective leptin signal transduction. Alternatively, insulin induction of suppressor of cytokine signaling 3 could inactivate the leptin receptor, through dephosphorylation of tyrosine 1138.[80] Figure 3 was adapted by J Kushner and is modified with permission from reference 80 © (2005) Lippincott, Williams & Wilkins. Abbreviations: 4E-BP1, eukaryotic translation initiation factor 4E binding protein 1; Akt, protein kinase B; eIF4E, eukaryotic initiation factor 4E; Foxo, forkhead box proteins; GRB2, growth factor receptor-bound protein 2; GSK-3β, glycogen synthase kinase 3β; IRS-2, insulin receptor substrate 2; JAK-2, Janus kinase 2; MAPK, mitogen-activated protein kinase; MEK, mitogen-activated extracellular-regulated kinase; mTOR, mammalian target of rapamycin; P, phosphate; $p70^{S6K}$, p70 S6 ribosomal kinase; PDK1, 3-phosphoinositide-dependent kinase 1; PI, phosphatidylinositol; PI3K, phosphatidylinositol 3 kinase; PKCζ, protein kinase Cζ; PTB, phosphotyrosine-binding domain; PTEN, phosphatase and tensin homolog; PTP-1B, protein-tyrosine phosphatase 1B; Raf, serine–threonine protein kinase; Ras; activator of Raf; S6, 40S ribosomal protein S6; SOCS-3, suppressor of cytokine signaling 3; SOS, son of sevenless; STAT3, signal transduction and transactivation 3; TSC, tumor suppressor C; Tyr, tyrosine.

CROSSTALK BETWEEN INSULIN AND LEPTIN

Acute insulin and leptin effects overlap

Insulin and leptin parallel one another's short-term effects in the CNS.[22] Both hormones are secreted during periods of energy sufficiency or excess, their receptors co-localize to the same VMH neurons, and both have similarly anorexigenic effects when administered directly into cerebrospinal fluid. Their similarities of action suggest they act as dual barometers of energy stores; insulin levels reflect short-term changes in energy intake, whereas leptin levels reflect energy balance over a longer period of time. Obesity is, however, a state in which the negative feedback that should result from chronic CNS exposure to

insulin and leptin is ineffective; appetite remains uncurbed and weight accrues despite adequate energy stores.

Chronic insulin antagonizes leptin signaling

Decreased central nervous system leptin transport

The observation that intracerebroventricular leptin is more effective than systemic leptin administration suggests that leptin resistance could be due to limited availability of the hormone in the CNS.[58] Indeed, low levels of leptin in cerebrospinal fluid have been documented in rodent models[59] and in human obesity.[60] Leptin is transported into the CNS via a transporter expressed in brain vasculature. This transport is decreased in obesity;[58] however, insensitivity to the behavioral effects of intracerebroventricular insulin and leptin has also been found in the chronically hyperinsulinemic obese Zucker rat strain, and also in DIO.[61] Inadequate transport into the CNS does not fully explain the leptin resistance seen in obesity, but may be a contributing factor.

Decreased neuronal leptin signal transduction

Although insulin and leptin bind to separate receptors, they share the IRS-2–PI3K signal transduction pathway.[18,22] Intracellular signaling by the leptin receptor is usually achieved through the Janus kinase 2–signal transduction and transactivation 3 (JAK-2–STAT3) pathway (Figure 3). Leptin's ability to activate STAT3 in hypothalamic neurons is reduced in DIO, which also induces hyperinsulinemia.[53] SOCS-3 and protein tyrosine phosphatase 1B are two proteins that regulate leptin sensitivity;[62] decreased expression (or knockout) of their respective genes enhances leptin sensitivity and gives rise to phenotypically lean mice. Studies of DIO and age-associated obesity suggest that SOCS-3 expression is affected in these states.[63] Insulin induces SOCS-3,[57] which then inactivates signal transduction from the insulin receptor, and inactivates the leptin receptor (by dephosphorylating tyrosine 1138).[56]

In addition, both insulin and leptin receptors use the low-abundance-message second-messenger IRS-2, in order to facilitate PI3K activity. The role of IRS-2 in leptin resistance is, however, controversial. One study suggested that knockout of IRS-2 in hypothalamic neurons promoted obesity and insulin resistance, and prevented leptin signal transduction, conferring a leptin-resistant state;[20] another study of IRS-2 knockout shows similar effects on promoting obesity, but with maintenance of leptin sensitivity.[21] Although not yet confirmed, these data suggest that hyperinsulinemia and resultant CNS insulin resistance may be a proximate cause of leptin resistance that promotes weight gain.

Adaptive advantage for insulin's role as a leptin antagonist

Teleologically, what could be the biological advantage for antagonism of leptin's actions by insulin in the obese state? Reversible antagonism of leptin's actions would be desirable in physiological phenomena in which rapid weight gain is essential. Similar conditions can be found during both puberty and pregnancy; two processes essential for reproduction. If leptin signaling could not be modulated, the weight accrual necessary for reproductive competence would be compromised. Indeed, both puberty and pregnancy are hyperinsulinemic and insulin-resistant states, because of the effects of sex hormones and growth hormone on insulin sensitivity in target tissues,[64] and of reciprocal increases in insulin release.

In both of these physiologic states, leptin levels increase sharply; postpuberty or postpartum, insulin levels fall, weight stabilizes or is lost, and leptin levels return toward the baseline state.[65,66] Insulin antagonism of leptin signal transduction is probably an integral control mechanism to ensure reproductive competence. In maladaptive conditions, however, when insulin rises chronically either through pancreatic hypersecretion (as is seen in hypothalamic obesity), or through insulin resistance (as is seen in certain ethnic populations, or in DIO in rodents), leptin signal transduction is impeded, and the obese state is either maintained or promulgated.

THE HEDONIC PATHWAY OF FOOD REWARD

The homeostatic pathway is not the only central arbiter of energy balance. Complementary to the ability of both insulin and leptin to alter feeding behavior, these hormones also modify the 'hedonic pathway' (which regulates pleasurable and motivating responses to stimuli). This pathway localizes to the ventral tegmental area (VTA) and the nucleus accumbens (NA), with inputs from various components of the limbic system, including the striatum, amygdala, hypothalamus and hippocampus. The hedonic pathway responds to drugs of abuse, such as nicotine and morphine. Food intake is responsive

to activation of the hedonic pathway; for example, administration of morphine to the NA increases food intake in a dose-dependent fashion.[67] Dopaminergic perikarya terminals project from the VTA to the NA, which mediates the rewarding and reinforcing properties of various stimuli, including food and addictive drugs. The VTA initiates feeding on the basis of palatability rather than energy need. Stimulation of this area triggers feeding behavior in rats that have already been fed, provided they are given a palatable food.

Insulin and leptin alter VTA–NA dopamine neurotransmission

Leptin and insulin receptors are expressed in the VTA, and both hormones have been implicated in modulating rewarding responses to food and other pleasurable stimuli. For instance, fasting or food restriction (during which insulin and leptin levels are low) increase the addictive properties of drugs of abuse, whereas intracerebroventricular leptin can reverse these effects.[68] In rodent models of addiction, increased addictive behavior, and pleasurable response from a food reward, as measured by dopamine release, is greater after food deprivation.[69] Obesity also results in decreased density of D_2 dopamine receptors as measured by PET scanning.[70]

In the short term, insulin increases expression and activity of the dopamine transporter, which clears and removes dopamine from the synapse; thus, acute insulin exposure blunts the reward of food in rats.[71] D_2-receptor antagonists and insulin act additively to acutely decrease the rewarding response to a palatable sucrose solution; furthermore, insulin seems to inhibit the ability of VTA-agonists (e.g. opioids) to increase intake of sucrose.[72] Finally, insulin blocks the ability of rats to form a conditioned place-preference association to a palatable food.[73]

Hyperinsulinemia could increase the reward derived from food

Insulin resistance at the VTA might contribute to obesity by preventing dopamine clearance from the NA, thus increasing pleasure derived from food in situations where energy stores are replete. CNS insulin resistance sets the stage for unchecked caloric intake in the face of positive energy balance, as evidenced experimentally by brain-specific insulin receptor knockout mice.[19] By altering hedonic responses to food, insulin resistance at the VTA may drive excessive energy intake.

WHERE DID THE HYPERINSULINEMIA COME FROM?

Hyperinsulinemia in the pediatric age group reflects genetic, epigenetic, and environmental origins. Firstly, children from certain ethnic groups have been identified to possess altered insulin dynamics compared with those of white, European ancestry, even before the development of obesity, which might predispose them to increased weight gain.[74] We have described increased insulin secretion among obese African Americans that is not explained by their obesity.[75] Secondly, the 'fetal origins of adult disease' hypothesis proposes that individuals who are either small-for-gestational-age or large-for-gestational-age at birth are prone to obesity and the metabolic syndrome in adulthood;[76] hyperinsulinemia and insulin resistance are present in both these birthweight states, and worsen with time.[77] Premature infants also develop insulin resistance in later life.[78] Indeed, low-birthweight babies who demonstrate the most rapid 'catch-up' growth after birth are those most likely to become obese.[79] Lastly, our current Western diet is highly insulinogenic, as demonstrated by its increased energy density, high fat content, high glycemic index, increased fructose composition, decreased fiber, and decreased dairy content.[80]

CONCLUSIONS: REINTERPRETING THE FIRST LAW OF THERMODYNAMICS

In this review, the CNS mechanisms underlying weight gain are described. Within the afferent arm of the homeostatic pathway of energy balance, leptin and insulin are often equated because of their short-term effects; however, the hyperinsulinemic state interferes with leptin signal transduction, as insulin acts as an endogenous leptin antagonist. This interference promotes leptin resistance, which should decrease SNS activity to reduce REE, and increase vagal activity to promote energy storage; except that, of course, the obese subject—of necessity—increases caloric intake, which raises their leptin concentration above the level at which leptin resistance occurs, so as to maintain normal energy expenditure and quality of life. The insulin resistance that is a characteristic of the obese state promotes further leptin resistance. Hyperinsulinemia also decreases dopamine clearance and uptake in the hedonic pathway, which promotes an increased reward of food that causes increased food intake. This effect also maintains the hyperinsulinemic state.

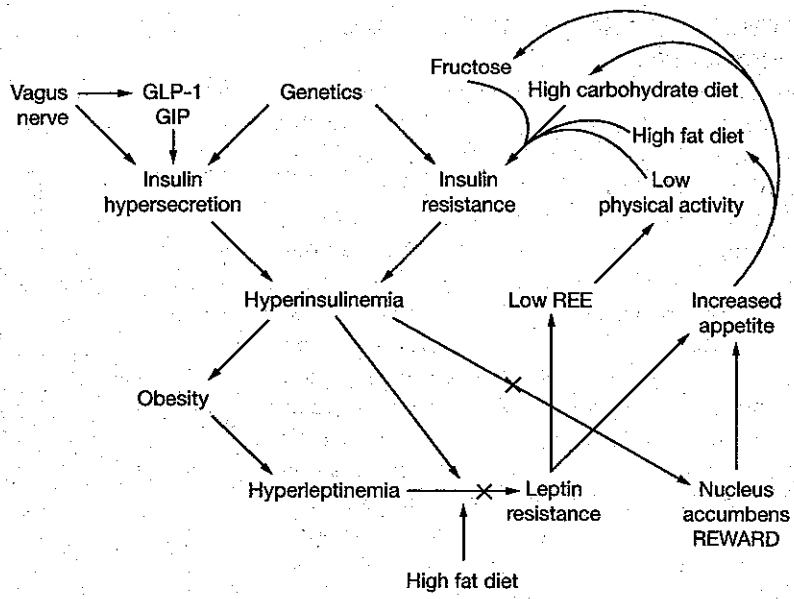

Figure 4 Insulin, leptin, reward and obesity. An algorithm describing the role of hyperinsulinemia in the dysfunction of the energy balance pathway. Various factors can lead to hyperinsulinemia, including vagus nerve mediated insulin hypersecretion, or hepatic and/or skeletal muscle insulin resistance. Hyperinsulinemia interferes with leptin signal transduction in the hypothalamus, promoting leptin resistance. This interference causes resting energy expenditure to decrease and appetite to increase, promoting further weight gain. In addition, hyperinsulinemia prevents dopamine reuptake at the nucleus accumbens, thus fostering increased reward associated with eating food, which promotes increased caloric intake. Hyperinsulinemia, therefore, converts the homeostatic and hedonic pathways from traditional negative feedback to feed-forward mechanisms.[80] Reproduced with permission from reference 80 © (2005) Lippincott, Williams & Wilkins. Abbreviations: GIP, gastric inhibitory polypeptide; GLP-1, glucagon-like peptide 1; REE, resting energy expenditure; X, inhibition.

The phenomenon of hyperinsulinemia turns two neuroendocrine negative feedback systems into feed-forward systems (or 'vicious cycle', in which the product stimulates further substrate production or action, rather than inhibiting it). Hyperinsulinemia promotes increased energy intake and decreased energy expenditure (Figure 4). Externally, this manifests as 'gluttony and sloth', but it is biochemically driven. In this paradigm, the First Law of Thermodynamics can be expressed so that the weight gain is primary, and the resulting behaviors are secondary: "If you store it (obligate weight gain occurs in response to hyperinsulinemia), and you expect to burn it (maintain normal REE and quality of life), then you must eat it (food intake increases)".

Although there are numerous contributors to hyperinsulinemia, our current food and activity environment is the most important, and the most amenable to change; however, it will take acknowledgment of the concepts of biological susceptibility and societal accountability, and de-emphasis of the concept of personal responsibility, to make a difference in the lives of children.

KEY POINTS

- Behavior has biochemical underpinnings, particularly the pathologic behaviors in disease states
- Obesity is characterized by hyperinsulinemia and leptin resistance
- In the long term, insulin functions as an endogenous leptin antagonist, it interferes with leptin signal transduction resulting in increased food intake and decreased physical activity
- Chronic hyperinsulinemia interferes with satiety by preventing extinction of the hedonic response to food
- Hyperinsulinemia has genetic, epigenetic, and environmental inputs

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Exhibit I

The New York Times
nytimes.com

March 4, 2008

Menu Fight Over Calories Leads Doctor to Reject Post

By STEPHANIE SAUL

A dispute over food industry influence has resulted in the resignation of the incoming president of the Obesity Society.

Dr. David B. Allison, who was to take over the society, a national group of obesity doctors and researchers later this year, submitted his resignation from that position on Friday.

Dr. Allison drew criticism from some members after he wrote an affidavit as a paid consultant on behalf of the restaurant industry, which is trying to block new rules in New York City that at the end of March will require fast-food and other restaurant chains to list the calories of menu items.

In an e-mail statement distributed to members of the organization last Friday, Dr. Allison apologized and said he would not serve as president partly because he was worried that he could not be effective in that role because of the affidavit.

"I truly regret and apologize for the distress it has caused the society," his e-mail statement said.

Dr. Allison's affidavit was filed in January in Federal District Court in Manhattan with a lawsuit filed by the New York State Restaurant Association against New York City. The affidavit said that providing calorie information could possibly have deleterious effects on obesity.

Because the position of Dr. Allison ran counter to the conventional thinking in his field, some critics contended that it illustrated the way industry money influences scientific and medical debate.

Dr. Allison did not reply to requests for comment.

Dr. Allison, a professor of biostatistics at the University of Alabama at Birmingham, has previously disclosed a long list of industry consulting relationships, including advisory roles for pharmaceutical companies and food companies like Coca-Cola, Kraft Foods and Frito-Lay.

In Friday's e-mail statement to society members, Dr. Allison acknowledged that he had been retained by lawyers for the New York State Restaurant Association to file the affidavit.

Dr. Allison said in the statement that he stood behind the scientific arguments in his affidavit, but he characterized his affidavit as a miscalculation in terms of Obesity Society politics.

"While I stand behind the scientific statements I made, my right to make them, and the manner in which I made them, I realize now that participating in this case while in the presidential sequence was a serious political error," he wrote.

Dr. Barry M. Popkin, director of the Interdisciplinary Center for Obesity at the University of North Carolina, Chapel Hill, said he believed Dr. Allison had resigned for the good of the organization. "I think this was a very difficult decision for David Allison," said Dr. Popkin, who had criticized Dr. Allison's decision to write the affidavit.

Dr. Popkin, a member of the group, said the organization was currently creating clear ethics guidelines for presidents and officers to help guide them.

Dr. Allison's affidavit had ignited a flurry of e-mail messages among obesity experts who questioned both Dr. Allison's position and his industry ties. It also led the Obesity Society to issue its own separate statement in supporting calorie labeling on menus.

"The Obesity Society believes that more information on the caloric content of restaurant servings, not less, is in the interests of consumers," said the statement by the group, which is based in Silver Spring, Md.

Dr. Allison had defended his role in the lawsuit, saying he was not arguing against implementation of the law, only presenting scientific evidence.

In the affidavit, Dr. Allison said that there was not sufficient evidence to show the effect of calorie labeling on people who eat in restaurants, and that while it was possible such labels on menus could be helpful, it was equally possible that they could have no effect — or that they might increase obesity, in part by casting high-calorie foods as forbidden fruit.

The proposal by the New York Board of Health has wide support from a broad group of organizations.

BRITISH Nutrition FOUNDATION

REVIEW

Labelling in restaurants: will it make a difference?

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Summary

The quest to solve the obesity epidemic in the United States has long been the centre of debate for health professionals, consumer and health advocates, scientists and politicians. While numerous suggestions have been made from these respective groups, there has been a steady increase, such that more than 64% of the US population are now classified as overweight or obese. The introduction of the Nutrition Facts label through the Nutrition Labelling and Education Act (NLEA) of 1990 has improved overall dietary quality, based on lower fat intakes and higher consumption of fruit and vegetables, in the 70–85% of the American population who use the label at least sometimes. More recently, however, concerns have been raised about the exemption of restaurants from the NLEA. The proportion of US consumers' food budget spent on food for consumption outside the home has increased from 26% in 1976 to 46% in 2004. It is also estimated that one third of a person's daily calories now come from food purchased for consumption outside the home. The United States Department of Agriculture and the US Department of Health and Human Services have jointly produced the 2005 Dietary Guidelines for Americans (DGAs) to provide evidence-based nutrition recommendations to the American public. Included are recommendations to limit the intake of sodium, cholesterol, saturates, *trans* fatty acids, alcohol and added sugars, to increase the intake of low-fat dairy products, fruit and vegetables, and to maintain energy balance. The food found in restaurants is abundant in nutrients recommended to be consumed in moderation, low in those that are recommended to be increased and higher in calories, when compared with a home-prepared meal. Given the consistent rise in obesity in the USA, the lack of food labelling in restaurants, the contrast between the nutrient recommendations of the DGAs and the nutrients found in restaurant food, and the increase in consumer spending on food purchased for consumption outside the home, suggestions now focus on passing legislation mandating food labelling in restaurants. Given the significance of such legislation to the US restaurant industry, this article highlights key considerations, as well as barriers, to restaurant food labelling. This article also emphasises the importance of conducting research to explore the impact of restaurant food labelling, prior to passing new legislation, as no studies have looked at the effect that this may have on restaurant patron behaviour. The importance of conducting research on restaurant labelling, prior to passing legislation, may be key to its success.

Keywords: food labels, legislation, Nutrition Labelling and Education Act, obesity, restaurants

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Introduction

The obesity epidemic that has overtaken the United States in recent decades is constantly in the limelight for health professionals, consumer and health advocates, and politicians. Obesity accounts for approximately 400 000 deaths annually in the USA (Mokdad *et al.* 2004) and costs the US economy an estimated 117 billion dollars per year (DHHS 2003). The health implications associated with obesity and overweight are well-documented, most notably the negative impact on heart health, type 2 diabetes and certain types of cancers. Nevertheless, more than 64% of the US population are obese or overweight (Flegal *et al.* 2002; DHHS 2003).

The gravity of the situation has been increasingly brought to the public's attention. The US Surgeon General delivered a Call to Action aimed at decreasing the incidence of overweight and obesity in 2001 (DHHS 2001). The 2005 Dietary Guidelines for Americans (DGAs) were published with specific nutrient recommendations and guidelines for the maintenance of energy balance (USDA 2005). The US Food Pyramid has been updated to 'MyPyramid' and is based on the 2005 DGAs (USDA 2005). The main changes made to MyPyramid are the tailored recommendations, taking age, sex and physical activity levels into consideration to provide people with specific nutrient and energy recommendations, rather than simpler, more general recommendations targeting the American population as a whole.

The implementation of food labelling, brought about by the Nutrition Labelling and Education Act (NLEA) of 1990 (FDA 1990), revolutionised the nutritional information available to American consumers. Studies indicate that, on average, 70–85% of the American adolescent, college and adult population read food labels at least sometimes (Marietta *et al.* 1999; Byrd-Bredbenner 2000; Burros 2004; Huang *et al.* 2004; O'Dougherty *et al.* 2006). Studies also indicate that people who use food labels typically have superior dietary quality, based on lower intakes of fat, and higher intakes of fruit and vegetables, when compared with those who do not use food labels (Kreuter *et al.* 1997; Kristal *et al.* 1998; Neuhouser *et al.* 1999; Perez-Escamilla & Haldeman 2002; Huang *et al.* 2004). While the positive impact that food labels have on the diets of those who use them has been demonstrated, there are several areas that do not fall under the jurisdiction of the NLEA. For example, restaurants are exempted from the NLEA. This is a cause for concern, due to the increased number of meals Americans currently purchase in food establishments compared with 1976.

Consumers' spending on food purchased for consumption outside of the home has increased from approximately 26% of their total food budget in 1976 to 46% in 2004 (Lin *et al.* 1999; Nestle & Jacobson 2000; Young & Nestle 2002; Kant & Graubard 2004; Wootan & Osborn 2006). The average American consumes approximately one-third of their calories from food purchased for consumption outside the home (Lin *et al.* 1999; Nestle & Jacobson 2000; Wootan & Osborn 2006). Eating in food service establishments can be problematic for numerous reasons. The specific nutrients and categories of foods that are outlined as causing problems in the 2005 DGAs are the nutrients and foods that are abundant in restaurant foods, such as sodium, cholesterol, total fat, saturates and *trans* fatty acids (Lin *et al.* 1999; McCrory *et al.* 1999; Nestle & Jacobson 2000; Kant & Graubard 2004; USDA/DHHS 2005; Wootan & Osborn 2006). Moderation in the consumption of these nutrients is recommended, and yet it has been demonstrated that these are the nutrients typically over-consumed by the American public (Lin *et al.* 1999; McCrory *et al.* 1999; Nestle & Jacobson 2000; Kant & Graubard 2004; Wootan & Osborn 2006). Similarly, foods and nutrients that are typically under-consumed and that should be increased, such as fruit and vegetables, fibre and calcium, are the nutrients least found in restaurant foods (Lin *et al.* 1999; McCrory *et al.* 1999; Nestle & Jacobson 2000; Wootan & Osborn 2006).

Furthermore, research has documented a positive association between eating out and bodyweight/percentage body fat (Wootan & Osborn 2006). This has been attributed to the higher energy content of restaurant foods when compared with home-prepared meals (Young & Nestle 2002; Kant & Graubard 2004). The cause of this is unclear; however, it has been linked to the accessibility of energy-dense foods and/or larger portion sizes in restaurants (Young & Nestle 2002; Kant & Graubard 2004).

Currently, nutritional information may be provided in restaurants on a voluntary basis; however, only 54% of the 300 largest chain restaurants in the United States provide such information, with most small chain restaurants and smaller establishments providing little or no nutritional information (Wootan & Osborn 2006). Furthermore, of the restaurants that make nutritional information available to consumers, 86% do so through the Internet, making it somewhat inconvenient for the consumer to access (Wootan & Osborn 2006). This method of conveying nutritional information does not enable consumers to make food choices at the point of sale based on the nutritional content of the food they are selecting. In contrast, making the information available

at the point of sale allows the consumer to compare food choices on the spot and make a more informed decision, as one does in the supermarket or grocery store when comparing products.

The US Food and Drug Administration (FDA) is the government agency that provides federal oversight of the current food labelling system. Given the emphasis placed on tackling the obesity epidemic by the US government, the FDA has been proactive in developing recommendations to help decrease the incidence of overweight and obesity in the USA. In 2003, the FDA formed the Obesity Working Group (OWG) in an attempt to provide evidence-based recommendations for the prevention of overweight and obesity. In 2004, the OWG published 'Counting Calories', a report that targeted, among other areas, the restaurant industry and its impact on obesity (FDA/CFSAN 2004). The OWG recommended that the FDA became more proactive and included restaurants in labelling requirements. However, making this change would require legislation passed by the US Congress and signed by the President. There are several considerations for the restaurant industry if food labels are mandated. These include the format of the label, the cost involved in its production, the need for nutrition professionals to be involved in the process, and the impact on restaurant sales. In order to address some of these issues, the OWG and the FDA recommend that more research be conducted to answer some of these questions.

The OWG and the FDA are not alone in their recommendations for labelling and nutritional information to be available in restaurants. Numerous health professionals, nutrition researchers, government agencies and politicians, respectively, have identified the need to implement labelling in food establishments. Despite the growing push for such legislation to be developed, and more importantly, the need for research in the area that has been identified, there has been no research demonstrating the impact that food labelling will have on consumer behaviour with respect to eating out. It has yet to be determined whether food labelling in the restaurant environment will lead to a change in the eating behaviour of US patrons. Furthermore, it is unknown whether restaurant labelling will have any impact on the incidence of obesity and overweight, the underlying reason behind the implementation of restaurant food labelling.

The current Nutrition Labelling and Education Act (NLEA)

The NLEA was passed by the US Congress in 1990 (FDA 1990). However, it was not until 1993 that food labels were required on food packaging. The US food label can be broken down into three parts: the nutritional information or nutrition facts panel, the ingredient list, and the nutrition or health claims. Current NLEA requirements stipulate that 15 items appear on the nutrition facts panel of food labels at all times, and that three additional nutrients appear in almost all cases (FDA 1990). The mandatory items include: serving size, servings per container, calories, calories from fat, the per cent daily value of each nutrient (a term derived from the reference daily intakes to aid consumers in making a healthy choice by showing them the percentage of the nutrient that is found in the food), total fat, *trans* fatty acids (*trans* fat), cholesterol (unless there is less than 2 mg per serving), sodium, total carbohydrate, protein, vitamin A, vitamin C, iron, and calcium (FDA 1990) (see Fig. 1). Originally, *trans* fatty acids were not required on the FDA label. However, given the research findings demonstrating the negative health effects of *trans* fatty acids, they were added to the list of required nutrients in January of 2006 (FDA 2003). The amount of dietary fibre and total sugars must be included if there

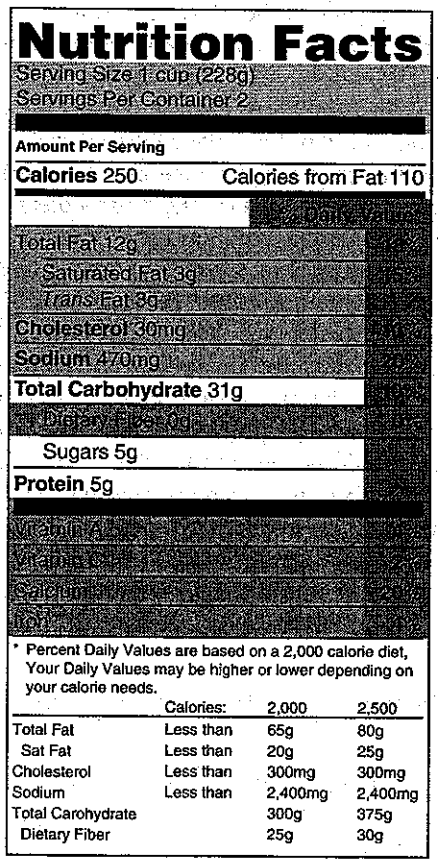

Figure 1 Nutrition facts. Source: FDA/CFSAN (2004).

is at least 1 g per serving of each nutrient, respectively, and the grams of saturated fat must be included if there is more than 0.5 g per serving (FDA 1990). All other nutrients may be put on the label, in accordance with the FDA format, on a voluntary basis. Nutrition and health claims must follow definitions provided by the FDA and can only be made when the claim can be supported by evidence. Furthermore, if there is a health claim made, then the nutrient that is referred to in the claim must appear on the label.

The NLEA makes several exemptions for food providers such that they are not required to provide food labels. Included in the exemptions are foods served or sold in restaurants, and foods that are served and sold for immediate consumption, *e.g.* in cafeterias, bakeries and delis. Although other exemptions exist, these two in particular permit restaurants to provide labels on a voluntary basis only. Despite the fact that those establishments exempted from labelling must support any nutrition or health claims with evidence, they are still not required to conduct any nutrient analysis of the product in question (FDA/CFSAN 2004). They are only required to provide the nutritional information specific to the nutrient involved in their nutrition or health claim, with respect to the FDA definition.

Given the increase in consumers' spending on restaurant food, it would be logical to conclude that the majority of the food purchased for consumption outside of the home would not include a label. Because research with respect to food labels on packaged foods has demonstrated a positive effect on the overall dietary quality in people who make use of food labels, it has been assumed that the same would apply if labelling was implemented in restaurants. Furthermore, given the nutrient content of foods sold in restaurants, and the health implications aforementioned of frequent dining in restaurants, health professionals, consumer and health advocates, and politicians have concluded that legislation should be passed to include mandatory food labelling in restaurants. US restaurant patrons might be more informed about what they are eating and thus their overall dietary quality would potentially improve, possibly mirroring the positive changes seen with the use of existing food labels.

2005 Dietary Guidelines for Americans (DGAs)

The 2005 DGAs were published jointly by the US Department of Agriculture and the US Department of Health and Human Services in an effort to provide Americans with evidence-based dietary advice. Given the wide array of organisations and sources that provide nutritional advice to the public, the US government recognised the need to voice their position on the subject. The 2005 DGAs contain many recommendations that are intended to moderate the intake of nutrients that are typically over-consumed and to increase nutrients that are typically under-consumed. Overweight and obesity, at a very fundamental level, can be attributed to a disruption in energy balance. Additionally, the intake of certain nutrients increases the risk for many chronic diseases, while the intake of other nutrients decreases the risk of certain diseases. Most notably, these diseases include hypertension, hypercholesterolaemia, cardiovascular diseases, type 2 diabetes and some types of cancer. These are also some of the most common chronic diseases associated with obesity and overweight.

The DGAs highlight the importance of energy balance by promoting the consumption of foods and beverages in amounts that meet an individual's estimated total energy expenditure (USDA/DHHS 2005). Specific recommendations emphasise the need to limit the intake of sodium, cholesterol, saturates, *trans* fatty acids, alcohol and added sugars (USDA/DHHS 2005). In contrast, it is recommended that the intake of fruit, vegetables and low-fat dairy products should be increased. As mentioned previously, restaurant food is high in the nutrients recommended to be consumed in moderation, high in calories, and low in the nutrients recommended to be increased in the diet.

Barriers to food labelling in restaurants

Implementing food labelling in restaurants poses problems on several levels for both the restaurant owner and the consumer. The first obstacles cited by restaurant owners are those that involve the menu (FDA/CFSAN 2004). There are several issues with respect to the menu, including variation, space and flexibility (FDA/CFSAN 2004). In many restaurants, changes to the menu occur frequently. Whether the items themselves change, or consumers request that a change be made to their order (*e.g.* extra sauce, no cheese, no mushrooms, extra bacon), there is a certain level of variability that cannot be accounted for. It is estimated that 70% of restaurant patrons request a menu modification for their food selection (Riehle 2003). There is also often a lack of standardised recipes in some places, making it difficult to determine nutrient content. Furthermore, due to marketing tactics, menu items change frequently and are concurrent with promotions (Wootan & Osborn 2006).

The requirement of food labelling would restrict this type of activity. The current NLEA label would require

a large amount of space on a menu or menu board. Additionally, the best format for a restaurant food label is debatable. It would be nearly impossible to include all the current NLEA requirements on a table menu or menu board. Restaurant owners who do not currently display labels have voiced this as being their primary concern with respect to labelling implementation (FDA/CFSAN 2004).

Another obstacle is the cost involved in producing food labels. Previously, a food label designed to meet NLEA requirements has been estimated to cost 500–655 US dollars (Boger 1995; FDA/CFSAN 2004). Given the input of nutrient values into software systems, the analysis of a food product has become much simpler and can now be undertaken without always requiring chemical nutrient analysis. Thus, the cost of food labelling has decreased and is now estimated to be 220 US dollars per label (Wootan & Osborn 2006). Due to some of the menu issues mentioned previously, it seems that having all food items on the menu analysed, based on current NLEA labelling requirements, would represent a substantial cost to restaurants. This barrier to food labelling was found to be more of an issue in restaurants that have not yet provided nutritional information on a voluntary basis (FDA/CFSAN 2004). American restaurant companies, especially larger chain restaurants, who have begun to analyse their products for the provision of nutritional information, have stated that once the cost of food labelling is incorporated into their budget and proper allocations have been made, this cost becomes less of an issue (FDA/CFSAN 2004).

A final barrier to labelling in the restaurant milieu involves employee training. The restaurant industry sees a large overturn of employees, and thus constant employee training is needed (FDA/CFSAN 2004). The implementation of food labelling in restaurants would require staff to have some knowledge of nutrition in order to answer any questions that may arise from customers. Restaurants implementing labelling would also need the advice of nutrition professionals to guide them on the development of healthier food choices and the labelling process itself. Furthermore, nutrition education for restaurant staff needs to be undertaken by a qualified professional and, therefore, the cost of conducting this training would increase (FDA/CFSAN 2004). A high employee turnover rate and a high cost of training would again increase the cost involved in implementing labelling in restaurants. Moreover, the demand for nutrition professionals would increase sharply, as dietitians and nutritionists would be needed to provide education to the restaurant industry, should labelling become mandatory (FDA/CFSAN 2004).

The restaurant industry

The US restaurant industry has recognised the increase in frequency of meals consumed outside of the home. The National Restaurant Association (NRA) conducts periodic surveys (every 4–6 years) of various components of the restaurant industry as a whole, and reported a 17% increase in the number of weekly meals consumed outside of the home from 1985 to 2000 (NRA 2001). The NRA also reported an increase in the amount of food dollars spent on meals bought for consumption outside of the home, and estimated that 46% of consumers' food budget goes towards purchasing these meals (NRA 2004). The US restaurant industry has responded to increased pressure for legislation obliging restaurants to provide nutritional information at the point of sale, by voicing concerns about menu obstacles (Riehle 2003). Nevertheless, research looking at nutritional information in restaurants indicates that labelling in restaurants is an attainable goal (Wootan & Osborn 2006).

Another concern raised by the restaurant industry is the perceived decrease in sales that might occur if food labelling was introduced. This was illustrated when the steakhouse style restaurant chain *Ruby Tuesdays* voluntarily provided food labels for their menu items (Fields & Sherman 2004). Their sales dropped markedly and, as a result, the labels were quickly removed from menus (Clark 2005). Conversely, competitor *Outback Steakhouse* saw a rise in their sales during the labelling period at *Ruby Tuesdays*. It could thus be speculated that the American public will not necessarily change their eating habits, but instead would prefer not to be confronted with nutritional information about what they are eating when choosing from a menu. In general, two-thirds of restaurants did not consider that labelling in their establishments would affect their sales (FDA/CFSAN 2004). However, of those who did consider it would affect sales, the majority believed that the effects would be negative (FDA/CFSAN 2004).

Conclusion

The amount of money spent on meals consumed outside the home has markedly increased in the USA. Numerous factors contribute to this, including the increased demand for convenience, the availability of food on-the-go, multiple-career households, as well as food and restaurant marketing tactics (Nestle & Jacobson 2000; Young & Nestle 2002; Kant & Graubard 2004). With an increase in eating outside of the home, there has been

an increase in intakes of some nutrients and a simultaneous decrease in others. Notably, increased amounts of total fat, *trans* fatty acids, saturates, total calories and sodium can be found in meals eaten outside of the home, while decreased intakes of fruit, vegetables, calcium and fibre are the norm (Lin *et al.* 1999; McCrory *et al.* 1999; Nestle & Jacobson 2000; Kant & Graubard 2004; Wootan & Osborn 2006).

Current US government nutritional recommendations have been modified to deliver a more tailored message to the public, identifying specific nutrients that need to be consumed in moderation, limited or increased. The nutrients recommended to be increased in the daily diet are those that tend to be lacking in restaurant food, while those that are recommended to be limited or eaten in moderation are the nutrients that are most abundant in restaurant-style food. Given the increase in overweight and obesity over the past few decades, there has been a quest to slow down this trend. While the US government and various other organisations have made numerous efforts to try and accomplish this, there has been no progress made in decreasing the proportion of the population that are overweight and obese in the USA. Numerous groups, including government agencies and professional associations, lobbyists, health professionals and researchers, have made several attempts to solve this persistent problem.

The US Surgeon General, the Institute of Medicine, researchers, government agencies, professional associations, health professionals, lobbyists and the American public have all called for legislation to make food labelling at the point of sale in restaurants mandatory (Almanza *et al.* 1997; Nestle & Jacobson 2000; FDA/CFSAN 2004; Hayne *et al.* 2004; O'Dougherty *et al.* 2006; Wootan & Osborn 2006). More and more advocates of restaurant labelling, including a growing number of state government officials, nutrition researchers, consumer advocates and professional organisations, are trying to achieve such legislation. However, there is a key point in this push that has not yet been addressed: will food labels in restaurants make a difference to consumer behaviour with respect to food choice? The answer is unclear, as there have been few published studies that have asked this question. A recent article suggests that 44–57% of college students would not be likely to look at food labels in restaurants (Krukowski *et al.* 2006). This same study found that 27–36% of the population studied could not accurately estimate their daily caloric needs. Although this may not be representative of the American population as a whole, it does raise concerns as to what impact, if any, amendments to include restaurants in the NLEA would have.

Many other questions with respect to food labelling in restaurants would arise in the event that they were included in the NLEA. These include the format that the label should take, the nutrients that should be included or not included, a comparison between food label use on packaged foods and foods eaten in restaurants, and what the impact would be on the general public in terms of their health. Therefore, many questions need to be addressed before an amendment to the NLEA is passed to include restaurants. Will food labelling in restaurants meet the ultimate goal of changing the way the public eats, resulting in healthier diets, healthier weights and a decrease in the incidence of chronic diseases? This key question is one that remains unanswered.

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Exhibit K

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NUTRITION LABELS AND OBESITY

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ABSTRACT

The Nutrition Labeling and Education Act (NLEA) imposed significant changes in the information about calories and nutrients that manufacturers of packaged foods must provide to consumers. This paper tests whether the release of this information impacted body weight and obesity among American adults. We estimate the effect of the new label using a difference-in-differences method. We compare the change before and after the implementation of NLEA in body weight among those who use labels when food shopping to that among those who do not use labels. In National Health Interview Survey data we find, among non-Hispanic white women, that the implementation of the new labels was associated with a decrease in body weight and the probability of obesity. Using NLEA regulatory impact analysis benchmarks, we estimate that the total monetary benefit of this decrease in body weight was $63 to $166 billion over a 20-year period, far in excess of the costs of the NLEA.

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Nutrition Labels and Obesity

The Nutrition Labeling and Education Act (NLEA) of 1990 represented the first comprehensive overhaul of the nation's food labeling laws in over half a century.[1] Prior to the NLEA, food labeling was voluntary and labels were required only for products containing added nutrients or that made nutrition claims. The NLEA, which took effect in 1994, made labeling mandatory for most processed foods. It requires manufacturers of packaged foods to display the Nutrition Facts panel, which lists in a standardized format the amount of key macronutrients, vitamins, and minerals contained per serving. The NLEA also requires manufacturers to use specified serving sizes within product categories, declare nutrients as a percent of the recommended Daily Value, and use approved health and nutrient content claims (Food and Drug Administration, 1999).

This paper examines whether the nutrition labeling changes introduced by the NLEA have impacted body weight and obesity among American adults. The NLEA was motivated by a growing body of scientific evidence linking dietary habits with obesity and chronic diseases such as cardiovascular disease, diabetes and some types of cancer. Two back-to-back reports documenting such evidence—the 1988 *Surgeon General's Report on Nutrition and Health*, and the 1989 National Research Council's *Diet and Health: Implications for Reducing Chronic Disease Risk*—provided impetus to the legislation. In 1990, the year the NLEA was enacted, voluntary labeling rules in existence were deemed seriously outdated with no nutrition information appearing on at least 40 percent of packaged foods and the available information "incomplete and misfocused," according to a study commissioned by the Food and Drug Administration (FDA) and the Food Safety Inspection Service (FSIS) (Porter and Earl, 1990). The new labeling regulations were expected to help consumers choose more healthful diets

through improved access to credible nutrition information (Kurtzweil, 1993, 1994). FDA's estimates of the health benefit from dietary changes triggered by the new labeling regulations ranged from $4.4 to $26.5 billion gained over 20 years. The total cost of NLEA implementation, including administrative and compliance costs, was projected to be between $1.4 billion and $2.3 billion (Food and Drug Administration, 1993).

The rising prevalence of obesity in the United States, however, has focused attention on the effectiveness of the NLEA labeling rules in improving health outcomes. The *Calorie Counts* report prepared by the FDA's Obesity Working Group in 2004 notes that "Despite reports of a positive correlation between label use and certain positive dietary characteristics, the trend toward obesity has accelerated over the past decade." Although obesity among Americans began to rise noticeably in the 1980s, its increase has continued unabated even after food labels were mandated by the NLEA. Data from the National Health Interview Survey (NHIS) suggests that adult obesity has increased continuously since the NLEA enforcement, rising from 18% in1995 to 23% in 2003. Other health surveys such as the National Health and Nutrition Examination Survey (NHANES) confirm this trend (Flegal et al., 2002). Aggregate adult medical expenditure attributable to obesity in 1998 has been estimated to range between $26.8 and $47.5 billion (Finkelstein, Fiebelkorn, and Wang, 2003).

The upward trend in aggregate obesity prevalence by itself, however, does not imply that the NLEA has been ineffective in stemming obesity. What exactly has been the effect of the mandatory nutrition labels introduced by the NLEA on the body weight and obesity among Americans? Surprisingly, despite being of considerable public policy interest, there has been

little empirical research on this question. The interest in better understanding the effect of food labeling on obesity and other dietary outcomes is not confined to the U.S. The World Health Organization's *Global strategy on diet, physical activity and health* suggests nutrition labeling as a way to meet the consumers' need for accurate, standardized and comprehensible information in order to make healthy food choices. A growing number of countries are implementing mandatory nutrition labeling regulations to achieve the public health goal of reducing obesity and preventing chronic disease (Hawkes, 2004).

In the next section, we review the existing literature on the effect of the NLEA and the Nutrition Facts panel information on different consumer outcomes. Section 3 describes our empirical strategy for estimating the effect of the new label on body weight and the data and specific measures that we use to implement this strategy. Section 4 reports our main findings and results of sensitivity analysis to check the robustness of the estimates. In section 5, we calculate the dollar value of the BMI reductions due to the NLEA and conclude with a discussion of the implications of our finding for mandatory nutrition labeling policy in the U.S. and other countries.

Previous Research

Although NLEA has been in effect for over a decade, relatively few studies have explored its effect on dietary intakes and, to the best of our knowledge, none has studied its effect on obesity.[2] A few studies in the nutrition literature have examined correlations or regression-adjusted associations between label use and dietary intakes and diet quality, and reported beneficial effects associated with label use (Kreuter et al., 1997; Neuhouser, Kristal, and

Patterson, 1999; Perez-Escamilla and Haldeman, 2002). A study that specified label use as an endogenous switching variable found significant beneficial effects for label use on the intakes of fats, cholesterol, sodium, and fiber (Kim, Nayga, and Capps, 2000). Their label use equation was identified by excluding a potentially endogenous variable—whether an individual considers nutrition an important factor when shopping for food—from the intake equations. Kristal et al. (2001) used data collected from two rounds of a Washington State consumer sample in 1995-96 and 1997-98 to predict the impact of label use on intake of fat and fruits and vegetables and found that use of food labels was associated with reduced fat intake. Despite the longitudinal data, the study did not control for unobserved effects that may have influenced both label use and intakes.

Moorman (1996) studied the effect of the NLEA on consumer comprehension and processing of label information using a longitudinal quasi-experimental design. The study gathered label use data from samples of consumers in October 1993 (N=554) and October 1994 (N=558)—eight months before and five months after the May 1994 implementation of the NLEA. Findings indicated that consumers acquired and comprehended more nutrition information following the introduction of the new labels. Relatively more information was acquired about nutritionally unhealthy products compared with healthy products, suggesting that labels may have a public health benefit. These findings are supported by another study with a similar pre- and post-NLEA design (Balasubramanian and Cole, 2002). NLEA changed consumer attention to negative nutritional attributes (such as fat and sodium, of which less is better) and more motivated consumers registered greater label search intensity in the post-NLEA period compared with the pre-NLEA period.

Overall, research on the relationship between food label use, particularly as implemented under the NLEA, and dietary and health outcomes remains limited (Institute of Medicine, 2003; Pappalardo, 2001). This is the case even though there has been a spirited debate about the NLEA's effectiveness, which has only sharpened in light of the obesity epidemic (Philipson, 2005).

Empirical Strategy and Data

Estimating the label effect

The main innovation in food labeling introduced by the NLEA is the Nutrition Facts panel, which presents essential nutrition information in a standardized, easily comprehensible format. Thus, much of the benefit from the NLEA was expected to occur when consumers read the labels and use the information in their food selection (Kurtzweil, 1994; Zarkin et. al., 1993). Labels existed before the NLEA under the voluntary labeling rules established by the FDA in 1975, but they were not on all packaged foods and were not standardized. We exploit this fact and estimate the effect of the new label on body weight by comparing the change in body weight of individuals who used labels in their food selection in periods before and after the NLEA's implementation with the change in the body weight over the same period of those who did not use the labels.

Suppose $\overline{WGT}$ denotes the mean of the body weight outcome variable, and that u and n indicate label user/nonuser status, and that a and b indicate periods after and before NLEA. Using the difference-in-differences (DD) strategy, the label effect δ_l can be estimated from the equation

(1) $$\delta_I = (\overline{WGT}_u^a - \overline{WGT}_u^b) - (\overline{WGT}_n^a - \overline{WGT}_n^b).$$

Ideally we would have data from a randomized experiment in which subjects were randomly assigned to either the label user (u) or the label nonuser (n) groups. Since our data is nonexperimental, selection bias is a potential problem. That is, nonusers may have had different changes in weight than label users over the period we examine for reasons that had nothing to do with the NLEA. To address this, we control for observable correlates of weight in the following regression:

(2) $$WGT_i = \beta_0 + \beta_1 LABEL_i + \beta_2 NLEA_i + \delta_I LABEL_i * NLEA_i + \beta_3 X_i + \beta_4 X_i * NLEA_i + \varepsilon_i.$$

The dependent variable WGT_i represents the weight outcome of the ith individual. $LABEL_i$ is an indicator variable that equals 1 if the individual is a label user and 0 if the individual is a label nonuser. $NLEA_i$ is an indicator variable that equals 1 for the post-NLEA years and 0 for pre-NLEA years. The coefficient δ_I for the $LABEL_i * NLEA_i$ interaction variable is the difference-in-differences (DD) estimate of the effect of the NLEA. The vector of characteristics X_i controls for differences in weight outcomes due to other influences, and the interaction terms $X_i * NLEA_i$ control for variation in the impact of characteristics across the pre/post-NLEA periods. These two sets of regressors (X_i and $X_i * NLEA_i$) are included to address the fact that our data are non-experimental. We assume that our treatment group indicator $LABEL_i$ is uncorrelated with the residual weight ε_i after we control for the observable correlates of weight X_i and $X_i * NLEA_i$. As we explain in the data section, these observable correlates of weight include age, marital status, education, income, family size, urban, region, smoking status, self-reported health, dieting behavior, and other information about diet.

Data Source

We implement this empirical strategy using data from the National Health Interview Survey (NHIS). The NHIS is a multipurpose survey of the U.S. civilian noninstitutionalized population fielded annually since 1958. A probability sample of households is interviewed weekly by trained Census interviewers to obtain information about the health and sociodemographic characteristics of the household members. Additionally, supplementary questionnaires are administered periodically to gather detailed information on specific health topics of interest. Approximately 36,000 to 47,000 households, including 92,000 to 125,000 persons are interviewed each year. NHIS is the principal source of health statistics in the U.S. (National Center for Health Statistics, 2000).

The NHIS uses a multistage probabilistic sampling design to represent the U.S. civilian noninstitutionalized population. Each person in the covered population has a known nonzero probability of selection. Sample weights provided in the NHIS data files reflect these probabilities of selection, along with adjustments for nonresponse and post-stratification. The empirical analyses in this study were undertaken taking into account the sample weights and the complex sample survey design. All estimates were generated using the SUDAAN software (Research Triangle Institute, 2005).

Nutrition Label Use

Our primary covariate of interest is the label use behavior of consumers over a time period that spans the NLEA implementation. During the 1991, 1993, 1995, and 1998 rounds of the NHIS, supplementary modules administered to adult respondents aged 18 and older included an

identically worded question on food label use: "When you buy a food item for the first time, how often would you say you read the NUTRITIONAL INFORMATION about calories, fat, and cholesterol sometimes listed on the label—would you say always, often, sometimes, rarely, or never?" As noted earlier, currently the nutritional information listing the amount of calories, fat, and other nutrients is found on the Nutrition Facts panel. Under the voluntary labeling regime that existed prior to the NLEA, up to 60% of packaged foods had some form of labeling listing such nutrients (Porter and Earl, 1990). The NHIS label use question is thus general enough to capture label use before and after the NLEA. For our analysis, we classified "Always/Often/Sometimes" responders into the label user group and "Rarely/Never" responders into the label nonuser group.

Since the NLEA was enforced starting May 1994, we consider the 1991 and 1993 waves of NHIS to be pre-NLEA data, and the 1995 and 1998 waves to be post-NLEA. Compared with previous studies such as Moorman (1996), the four NHIS samples provide a broader span of time around NLEA's implementation to assess its impact. The 1993 and 1995 samples provide data immediately prior to and following the NLEA and the 1991 and 1998 samples provide data more separated from the event. Our main empirical estimates are derived from pooled data with an indicator for pre-NLEA and post-NLEA periods. Unlike previous studies, the large sample size provided by the NHIS enables us to conduct analysis by race/gender subgroups. This is particularly important since weight outcomes have a significant genetic component and cultural/gender factors exert a strong influence on weight-related behaviors (Cawley, 2004).

In addition to the five categories of frequency of label use ("Always" to "Never"), respondents were given a sixth option of "Don't buy food." Those who don't buy food are neither in the treatment nor control group and so we exclude them from our main models. (After discussion of our primary results, we explain how our results change if we include these individuals in our sample.)

Table 1 reports the proportions of the population who report reading labels when buying food. Estimates for pre- and post-NLEA periods as well as 1991 and 1998 are presented, both for the overall adult population and broken down by race/gender groups. Overall label use is steady over the pre- and post NLEA periods at 67%, although there is slight increase of 0.7 percentage-points going from 1991 to 1998. In the way of comparison, using a similar label use definition as ours, FDA's Health and Diet Survey registered 70% label use in 1994 and 69% in 1995 and 2002 (FDA, 2004). In short, the NLEA does not seem to have altered the percent of people who use labels.

The proportion of those who report that they don't buy food rises from 4% in 1991 to 8.5% in 1998. A greater proportion of men than women report not buying food. For example, in the post-NLEA period, 11.8% of white men report not buying food compared to 2.1% of white women. In absolute terms, the increase in not buying food is mainly among men, although the proportion of women not buying food also increased from 1991 to 1998. One reason for the increase in the proportion of those who don't buy food could be an increase in the number of individuals almost exclusively dining out.

Body Weight and Obesity

We used Body Mass Index or BMI (ratio of weight in kilograms to the square of height in meters) as our primary outcome measure for estimating the impact of the new label on body weight. BMI was calculated from self-reported height and weight. In 1998 NHIS, all respondents personally reported their height and weight. In the NHIS for 1991, 1993, and 1995, height and weight were provided mostly by respondents, but in some cases proxy reports were used. NHIS provides a flag for proxy-reported height and weight and we excluded all such cases from our analysis. Since the onset of the health effects of excess body weight are especially linked to when a person is obese, we used an indicator for obesity status, defined as BMI $\geq$30, as an additional outcome measure.

Table 2 reports the mean BMI and percentage obese estimated for different population groups. In general, both the mean BMI and the prevalence of obesity have increased over the pre- and post-NLEA period. Obesity is highest at around 30% among black women. Although white women have the lowest obesity, they recorded a higher rate of increase both in the pooled and yearly pre- and post-NLEA data compared with black women.

Other Covariates

The goal of our empirical analysis is to estimate NLEA's impact by comparing the change in body weight of label users over the pre/post-NLEA period with the change in body weight of label nonusers over the same period. NHIS respondents are not randomized into the treatment group (label users) or control group (label nonusers); they self-select into these groups. Therefore, label users and nonusers are likely to have different characteristics, and it is important

to control for these differences. Previous research suggests that income, education, age, gender, household size and urban residence have significant influence on label use behavior (Kim, Nayga, and Capps, 2000; Neuhouser, Kristal, and Patterson, 1999). Table 3 lists these, as well as several additional sociodemographic and health characteristics that are included as controls in our models. There are some notable differences between the pre- and post-NLEA samples, which confirms the need to control for these variables in our empirical models. Distribution of family income has shifted lower in the post-NLEA period. The NHIS provides only a categorical income measure. The original measure has a significant number of missing values. NHIS data files include separate family income files which replace the missing values with imputed incomes. We used these income measures in our analysis. For 1998, family income was imputed using a multiple imputation procedure. We took this into account using SUDAAN's multiple imputation option.

In 1993 and 1995, the supplementary questionnaires containing the label use questions were administered to only half the sample of adults. In 1995, these adults were distributed evenly throughout the year. However, in 1993, the half sample consisted of adults interviewed in the second half of the year. This is reflected in the higher proportion individuals in the 3rd and 4th quarters in the pre-NLEA sample (table 3). Any seasonality introduced by this is taken into account by including quarter dummy variables in our models.

We included several covariates to account for possible differences and changes in health status, health behavior, and health attitudes. Health condition and health preferences were controlled by including self-assessed health status, number of days bedridden in the past 12 months, smoking

status, and a variable indicating the frequency of adding salt to food at the table. Preferences for weight were proxied using indicators for individual's current action regarding his or her body weight (e.g. trying to lose weight, trying to gain weight, not doing anything).

Results

Basic Results

Table 4 reports estimates of the terms in equation 1, with the DD estimates of the NLEA effect, $\hat{\delta}$, presented in the final column. These estimates in Table 4 reflect models that do not control for sociodemographic or health characteristics (we will later present estimates from models that do control for those characteristics). The upper panel presents results based on BMI and the bottom panel gives results based on percent obese. Estimates were obtained for the overall population and for four major non-Hispanic race/gender subgroups. In all cases, label users had greater body weight compared with label nonusers in the pre-NLEA period. For example, the 1991-93 obesity prevalence in the population overall was 17.07% among label users and 14.51% among label nonusers. With the exception of white women, this pattern held in the post-NLEA period as well. This is likely because, at any given time, heavier persons may be using labels in their attempt to lose weight. For this reason, differences in weight status between label users and nonusers at any given time cannot be used to draw inference on the efficacy of the labels. What we are interested is in the change in weight status of label users and nonusers and the difference between these changes. These estimates are reported, respectively, in columns 3, 6, and 7.

Column 3 estimates in table 4 show that in most cases, the body weight of label users increased significantly from the pre-NLEA period to the post-NLEA period. Thus, clearly the effect of the

new labels hasn't been forceful enough to reverse the trend of rising obesity. However, besides label information, many other economic and social factors influence body weight. Changes in these factors could mask any potential label effect. Since these factors affect label nonusers as well, an estimate of their effect on body weight is given by the difference in weight of label nonusers across the pre- and post-NLEA periods. These estimates reported in column 6 show that, with the exception of black men, body weights of label nonusers also increased significantly from the pre-NLEA to the post-NLEA period. Column 7 reports the DD label effect, that is, the change in weight of label users relative to that of label nonusers. With non-label effects removed, the body weight of the overall population of label users actually fell in the post-NLEA period, as measured both by the mean BMI and by percent obese. However, breakdown of the label effect by race/gender groups reveals great disparity. The largest beneficial label effect on body weight is for non-Hispanic white women. With the introduction of the new labels, there was a net reduction of 0.52 kg/m^2 in their BMI and a 3.36 percentage-points reduction in obesity. Obesity declined among black women as well, by nearly 5 percentage points, although the decline in their BMI wasn't statistically significant. New labels had no effect on the body weight of white men. For black men, we get a seemingly anomalous result: introduction of new labels actually increased their body weight. However, before reading too much into these results, we have to account for the possibility that the results could be due to selection bias—difference in sociodemographic characteristics between label users and nonusers. To control for such differences, we adopt the regression framework in equation 2.

Regression Results

Table 5 presents label effects estimated from DD regressions with BMI as the dependent variable. We estimated two regression models, one with a limited covariate specification that included only the sociodemographic characteristics and a second one with a full covariate specification that included the sociodemographic characteristics plus the health characteristics. These health characteristics are endogenous. For example, some individuals may smoke because smoking acts as an appetite suppressant and thus help them to reduce their weight. Those reported to be in excellent health may be so because of lower body weight. And those trying to lose weight are likely to do so because they perceive themselves to be overweight. However, these variables are also likely to capture individuals' preference for health and attitudes toward body weight that may affect their current weight and change in weight over time. Therefore, we estimated a separate model with these health variables included.

In both the limited and full models, characteristics were allowed to have period-specific effects on BMI through interactions between the pre/post-NLEA indicator and the covariates. Both models were estimated for the overall population and by non-Hispanic race/gender groups. For brevity, only the estimated label effects—that is, the coefficient of the $LABEL_i * NLEA_i$ interaction, δ_1—are reported.

The results are fairly clear. Non-Hispanic white women benefited from the new food labels introduced by the NLEA. Based on the estimate from the full covariate specification, their BMI on average is 0.3 kg/m^2 lower than it would have been without the new labels. The label effect is insignificantly different from zero for the rest of the race/gender groups and for the overall population, under the full covariate specification. For black men, the anomalous increase in

weight is observed—but only in the limited covariate specification. With health variables included under the full covariate specification, the magnitude of the label effect for black men is reduced nearly by half and the estimate is statistically insignificant.

The inclusion of health variables in the model adds considerable explanatory power to the BMI regressions; in some cases the R^2s triple. The effects of covariates are along the expected lines (estimates not reported here but are available from the authors). Based on the regression estimate for the overall population, *ceteris paribus*, smokers have significantly lower BMI than non-smokers. Those reported to be in excellent or very good health have, on average, 1.5 kg/m^2 lower BMI compared with those in fair or poor health. Those who are trying to lose weight have 3.5 kg/m^2 higher BMI and those who are trying to gain weight have 3.7 kg/m^2 lower BMI compared with those who are trying to stay the same or not doing anything about their weight. All the included sociodemographic variables have significant correlations with the BMI. BMI is positively related to age and negatively to family income and individual's education level.

Table 6 reports DD estimates of the label effect with a person's obesity status as the dependent variable. We report results from a linear probability model (with coefficients converted to percentage points by multiplying by 100). Results from logistic regressions were similar. The obesity regressions lead to the same conclusions as the BMI regressions: Non-Hispanic white women benefited from the introduction of the new labels. Based on the full covariate specification, obesity prevalence among white women is 2.36 percentage-points lower than it would have been without the new labels. The labels have a statistically insignificant effect on obesity prevalence among the other race/gender groups and for the population overall. Once

again, labels seem to be associated with an increase in obesity among black men, but this effect is insignificant in the full model.

Both the basic and regression results discussed so far were from samples that excluded adults who responded "Don't buy food" to the label use question. We reestimated the BMI and obesity regressions models reported in tables 5 and 6 with these individuals included in the label nonuser category. The results were similar, with a consistent beneficial effect found for non-Hispanic white women. The results were very stable under the limited covariate specification—label effect on BMI was –0.4 for white women, the same as in table 5. In the full covariate specification, the estimates were slightly lower and less precise. This instability is surprising given that among white women, those who don't buy food constitute a very small fraction, 2.1% in the post-NLEA period. This suggests that non-food-buyers are a special group that should be excluded from the comparison group as was done for models reported in tables 5 and 6.

Effects Across Different Periods

A key assumption in interpreting the DD regression results is that the label effects estimated across the pre-/post-NLEA periods are due to the enforcement of mandatory labeling in 1994 and not due to an underlying trend in weight change that already existed before the introduction of the new labels. With two years of pre-NLEA data (1991 and 1993) and two years of post-NLEA data (1995 and 1998), we can test this assumption. Table 7 reports DD label effects for non-Hispanic white women estimated across different time periods. The key results appear in the first row. This row gives the change in BMI and obesity due to label use from 1991 to 1993. The unadjusted results are positive and significant while the covariate-adjusted effects are also

positive but not significantly different from zero. This suggests that there was no pre-existing trend in the weights of white women label users compared with white women label nonusers. If anything, the underlying trend for label users relative to label nonusers in the pre-NLEA was of weight gain rather than weight loss. This trend reversed sharply in the post-NLEA period, as the rest of the results in table 7 show. Between 1993 and 1995, the label effect resulted in a reduction of 0.45 kg/m^2 of BMI among non-Hispanic white women. Comparing 1993 with 1998, obesity fell by 3.2 percentage points.

Effects Across BMI Distribution

The results with obesity as the dependent variable show that label effects for non-Hispanic white women occur at parts of the BMI distribution that matter most for health outcomes—those who are heaviest. To illustrate this more clearly, we estimated the basic DD label effects at various points along the BMI distribution. Table 8 reports these results, which are not adjusted for covariates. The label effect at the median, -0.5 BMI units, is essentially the same as at the mean (-0.52) reported in Table 4. The label effects, however, are larger at the upper end of the BMI distribution than at the lower end, as the estimates charted in Figure 1 clearly show. These results confirm that the benefits of the label are concentrated among non-Hispanic white women of higher body weights.

Monetary Impact of the NLEA

Prior to the implementation of the NLEA, the FDA had estimated the health benefits from the new labels to be between $4.4 and $26.5, realized over 20 years (Food and Drug Administration, 1993). This estimate can be put in perspective given our finding of a beneficial effect of the

label for non-Hispanic white women. We translate our estimate of the average reduction in BMI of white women due to the new label into dollar terms using four sources of potential benefits from the BMI reduction: lower mortality risk, lower medical expenditures, reduced absenteeism, and increased productivity.[3] Estimates of the implied marginal benefit from these sources associated with a reduction in one unit of BMI are obtained from published sources. We then assess the per-person value of the benefit attributable to the new label using our preferred estimate of the BMI reduction of 0.3 due to the NLEA (table 5). For each source, this estimate is then multiplied by the number of non-Hispanic white female label users to get the total benefit attributable to the new label from that source. The number of non-Hispanic white women label users is estimated by multiplying the census estimate of white women in 1994 (the year of NLEA implementation) by 0.75, the average fraction of white women label users in our data. Finally, the benefits from all four sources are summed to obtain the total benefits from the NLEA.

Table 9 reports our monetary benefit estimates. The estimate for the reduction in mortality risk associated with lower BMI is from Calle et. al. (1999). They report age-adjusted death rates for white females at different BMI ranges. Based on overweight prevalence estimates reported by Flegal et al. (1998) using the 1988-94 NHANES data, we assume that the median non-Hispanic white female BMI in 1994 to be in the 23.5-24.9 range. From Calle et al., the implied marginal reduction in death risk for a 1-unit decline in BMI for white women from this range to the next lower category is 0.0133 percentage points. Using FDA's (Zarkin et. al., 1993) estimate of the value of statistical life in 1988 dollars ($1.5 million), the 0.3-unit BMI decline resulting from the NLEA has a monetary benefit of $60 per white female label user.[4] Across all white female label users, this implies that the reduction in mortality associated with the NLEA is worth $4.4 billion

per year. In its final regulatory impact analysis for the NLEA, FDA reported the value of health benefits gained over a twenty-year period, in 1991 dollars, using a 5-percent discount rate. In the final column of table 9, we report comparable figures for each source of health benefits. The estimated value of benefits from mortality risk reduction associated with label use among non-Hispanic white women over 20 years is $63 billion.

Similar calculations for benefits from the decrease in BMI associated with the NLEA imply benefits of about $23 billion from reduced medical expenditure (based on estimates for females from Finkelstein, Fiebelkorn, and Wang, 2003), $ 8 billion from lower absenteeism (based on estimates for females Finkelstein, Fiebelkorn, and Wang, 2005), and $71 billion from increased productivity (based on estimates for white females from Cawley, 2004).[5] Including mortality, the total benefits from the NLEA gained over a 20-year period is $166 billion (in 1991 dollars), which is far higher than the upper limit of both the costs ($2.3 billion) and the benefits ($26.5 billion) projected by the FDA. If mortality benefits are excluded, the benefits over 20-years are $102 billion. Based our estimates, therefore, the costs of the NLEA are far exceeded by the benefits associated with the reduction in the body weight of non-Hispanic white women.

Discussion

The Nutrition Labeling and Education Act (NLEA), enacted in 1990 and enforced since 1994, represented the first comprehensive overhaul of U.S. food labeling laws in 50 years. By providing access to consistent, standardized, and credible nutrition information, the law was expected to help consumers choose more healthful foods and thereby promote better health outcomes. FDA estimated the value of health benefits from new labels to be between $4.4 and

$26.5 billion, realized over 20 years. The total cost of NLEA implementation, including administrative and compliance costs, was projected to be between $1.4 billion and $2.3 billion.

To date, there has been little evaluation of NLEA impact dietary and health outcomes among Americans. Using a unique set of pre- and post-NLEA data on the label use habits of U.S. adults, this paper examined whether mandatory labeling introduced by the NLEA had an impact on body weight. The findings indicate that the NLEA labels had a beneficial impact, but only for one demographic group—non-Hispanic white females. As a result of the new labels introduced by the NLEA, the BMI and probability of obesity among white female label users were significantly lower than they would have been in the absence of the new labels. The total monetary benefit due to lower mortality, reduced medical expenditures, declining absenteeism, and increased productivity associated with this reduction in body weight was estimated to be about $166 billion (1991 dollars) over a 20-year period.

Our estimates have significant implications for nutrition labeling initiatives in the U.S. and at the international level. One important implication is that label benefits may be limited to certain demographic groups. Future research should investigate why most groups in the U.S. do not seem to benefit (at least in terms of body weight) from these regulations. A second implication is that our assessment of the benefits of the mandatory labeling initiative in the U.S. may offer guidance for similar initiatives being considered by other countries.

Our study has some limitations. First, it is based on non-experimental data. If our controls (which include time-varying effects of education, income, marital status, health, health

behaviors, and dieting attempts) do not adequately capture how self-selection among label users may have changed over time, our estimates could be biased. However, examining the stability of our results against two sources of such bias—the changing effects of unobservables using their observable correlates and testing for the existence of an underlying trend in the body weight of label users and nonusers in years prior to NLEA—suggests that any bias is likely to be small. Second, there may be additional benefits due to dietary changes associated with the increased supply of healthier foods from industry reformulation of foods following the NLEA. Such benefits are not taken into account in this study. Third, any improvements in consumer welfare due to substitutions among foods that may result from the release of new nutrition information following NLEA's implementation are also not considered in this study.

Footnotes

1. For a timeline of food labeling laws in the United States and events leading up to the NLEA, see Kurtzweil (1993).

2. There is a more extensive literature on the determinants of label use, on the effect of nutrition labels that predate the NLEA on dietary outcomes, and on NLEA's impact on the food manufacturing industry. See Porter and Earl (1990), Teisl and Levy (1997), and Variyam (2005) for examples.

3. FDA's benefits estimates were based on the number of life-years gained due to dietary changes triggered by the new label. Specifically, the benefits were assumed to result from lower intakes of total fat, saturated fat, and cholesterol and the associated reduction in the risk of coronary heart disease and cancer (Zarkin et al., 1993). Therefore, our result for benefits from the reduction in mortality risk is the one most comparable to FDA estimates.

4. Since overweight and obesity likely increased between 1988 and 1994, the assumption of median BMI in the 23.5-24.9 range for non-Hispanic white women in 1994 is conservative. If median BMI in the 25.0-26.4 is assumed, the per-person value of the implied marginal reduction in mortality risk increases to $162.

5. For medical expenditure and absenteeism, the implied marginal benefits are derived from a shift from the obese to the overweight category. For productivity, Cawley (2004) provides an estimate of the rise in wages that would result from a 1-unit reduction in BMI for white females. We interpret the wage as the marginal revenue product of labor, and therefore as a measure of productivity.

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Table 1. Nutrition Label Use in pre- and post-NLEA periods, 1991-98 NHIS

Population	1991-93	1995-98	1991	1998
Overall				
Excluding "Don't Buy Food"	67.0	67.1	66.1	66.8
Including "Don't Buy Food"	64.7	62.5	63.4	61.1
Don't Buy Food	3.4	6.8	4.0	8.5
Non-Hispanic White Men				
Excluding "Don't Buy Food"	57.8	58.8	57.3	58.8
Including "Don't Buy Food"	54.0	51.9	52.9	50.5
Don't Buy Food	6.6	11.8	7.6	14.1
Non-Hispanic Black Men				
Excluding "Don't Buy Food"	58.3	57.7	58.8	59.9
Including "Don't Buy Food"	54.1	49.7	53.9	49.5
Don't Buy Food	7.3	13.8	8.3	17.4
Non-Hispanic White Women				
Excluding "Don't Buy Food"	75.2	76.8	73.8	76.7
Including "Don't Buy Food"	74.3	75.2	72.7	74.9
Don't Buy Food	1.3	2.1	1.5	2.4
Non-Hispanic Black Women				
Excluding "Don't Buy Food"	70.4	68.5	69.1	69.5
Including "Don't Buy Food"	69.6	66.4	68.0	67.0
Don't Buy Food	1.1	3.1	1.6	3.6

Table 2. Body Mass Index and Obesity in pre- and post-NLEA periods, 1991-98 NHIS

Population	1991-93	1995-98	1991	1998
		BMI (kg/m^2)		
Overall	25.5	26.2	25.4	26.3
Non-Hispanic White Men	26.1	26.6	25.9	26.7
Non-Hispanic Black Men	26.5	27.3	26.3	27.6
Non-Hispanic White Women	24.8	25.4	24.6	25.5
Non-Hispanic Black Women	27.6	28.1	27.4	28.2
		Obese (%)		
Overall	16.1	19.1	15.3	20.0
Non-Hispanic White Men	14.9	18.1	14.1	19.1
Non-Hispanic Black Men	18.1	22.9	16.0	25.7
Non-Hispanic White Women	14.8	17.6	14.3	18.0
Non-Hispanic Black Women	30.9	30.3	29.3	32.1

Table 3. Variables and sample means

Variable	1991-93	1995-98
Age 18-24	10.5	11.0
Age 25-34	22.6	21.0
Age 35-44	21.4	22.4
Age 45-64	26.5	28.5
Age 65+	19.0	17.1
Male	39.4	42.6
Female	60.6	57.4
Non-Hispanic White	77.0	75.5
Non-Hispanic Black	11.2	11.0
Non-Hispanic Other	3.9	4.1
Hispanic	8.0	9.5
Married	61.5	59.9
Widowed	9.3	7.8
Divorced/Separated	12.3	11.7
Never Married	17.0	20.6
Family income <$5,000	0.8	2.3
Family income $5,000 - $9,999	1.9	4.2
Family income $10,000 - $14,999	2.5	4.9
Family income $15,000 - $19,999	2.4	4.7
Family income $20,000 - $24,999	9.8	8.5
Family income $25,000 - $34,999	16.1	14.8
Family income $35,000 - $44,999	12.1	11.6
Family income > $49,999	54.5	48.9
Family size	2.6	2.7
	(0.01)	(0.01)
Less than High School (<12 yrs)	20.0	17.7
High School (=12 yrs)	37.0	32.4
Some College (13-15 yrs)	22.1	26.4
College (16+ yrs)	20.8	23.5
MSA	78.1	79.2
Non-MSA	21.9	20.8
Northeast	20.5	19.4
Midwest	24.6	24.7

Table 3. Variables and sample means

Variable	1991-93	1995-98
South	32.8	34.0
West	22.1	19.9
Quarter 1	11.6	25.0
Quarter 2	12.3	24.6
Quarter 3	38.1	25.3
Quarter 4	37.9	25.0
Read nutrition label always/often/sometimes	67.0	67.1
Read nutrition label rarely/never	33.0	32.9
Smoker	25.6	24.1
Nonsmoker	74.4	75.9
Excellent/Very Good Health	61.0	63.2
Good Health	25.7	24.9
Fair/Poor Health	13.3	11.9
Trying to Lose Weight	33.2	33.6
Trying to gain Weight	3.5	3.5
Stay the same/not doing anything	63.3	62.9
No. of bed days in past 12 months	6.1	5.0
	(0.2)	(0.2)
Always/often add salt to food at the table	22.9	22.3
Sometimes add salt to food at the table	20.9	23.1
Rarely/Never add salt to food at the table	56.1	54.6
Sample size (N)	64,760	49,757

Table 4. Mean BMI and Percent Obese by Label Use Status and the Estimated Difference-in-Differences Label Use Effect, 1991-93 / 1995-98

Population	Label User			Label Non-User			
	1995-98	1991-93	Δ_u	1995-98	1991-93	Δ_n	$\hat{\delta}=\Delta_u-\Delta_n$
				BMI (kg/m^2)			
Overall	26.26	25.67	0.59***	26.03	25.22	0.81***	-0.22**
Non-Hispanic White men	26.93	26.39	0.54***	26.28	25.73	0.55***	0.01
Black men	27.94	26.71	1.23***	26.61	26.17	0.43	0.79*
White women	25.44	24.92	0.52***	25.36	24.32	1.04***	-0.52***
Black women	28.53	28.13	0.40**	27.32	26.47	0.86**	-0.45
				Obesity (Percent)			
Overall	19.69	17.07	2.62***	18.40	14.51	3.89***	-1.27*
Non-Hispanic White Men	19.57	15.89	3.68***	16.52	13.89	2.64***	1.04
Black Men	26.60	18.92	7.67***	18.92	18.18	0.74	6.93**
White Women	17.29	15.18	2.11***	18.75	13.28	5.47***	-3.36***
Black Women	34.02	34.16	-0.14	28.16	23.32	4.84**	-4.98*

***=p<0.01, **=p<0.05, and *=p<0.10.

Table 5. Estimated Label Use Effect from Difference-in-Differences Regressions for BMI

Population	1991-93 / 1995-98				
	(1)		(2)		
	$\hat{\delta}$	R^2	$\hat{\delta}$	R^2	N
Overall	-0.09 (0.93)	0.08	-0.02 (.24)	0.23	85,753
Non-Hispanic White Men	0.06 (0.45)	0.06	0.16 (1.23)	0.22	24,186
Black Men	0.83** (2.04)	0.06	0.46 (1.25)	0.30	3,647
White Women	-0.40** (2.47)	0.06	-0.30** (1.99)	0.21	38,224
Black Women	-0.31 (0.81)	0.07	-0.20 (0.56)	0.24	7,615
Sociodemographic Variables included	Yes		Yes		
Health Variables Included	No		Yes		
Interactions of Included Variables and pre-/post NLEA indicator	Yes		Yes		

Absolute t-ratios are in parentheses; ***=p<0.01, **=p<0.05, and *=p<0.10. Here, and in all regressions, sociodemographic variables included are age, marital status, income, education, family size, MSA, region, and interview quarter. The health variables included are smoking, self-assessed health status, current effort to lose/gain weight, number of days bedridden in the past 12 months, and use of table salt. See Table 1 for definition of these variables.

Table 6. Estimated Label Use Effect from Difference-in-Differences Regressions for Obesity

Population	1991/93-1995/98				
	(1)		(2)		
	$\hat{\delta}\times100$	R^2	$\hat{\delta}\times100$	R^2	N
Overall	-0.36	0.03	-0.13	0.11	85,753
	(0.52)		(0.19)		
Non-Hispanic White Men	1.32	0.03	1.82	0.12	24,186
	(1.15)		(1.60)		
Black Men	6.68*	0.04	3.50	0.21	3,647
	(1.91)		(1.06)		
White Women	-2.67**	0.03	-2.36**	0.09	38,224
	(2.33)		(2.11)		
Black Women	-3.77	0.05	-2.07	0.17	7,615
	(1.34)		(0.77)		
Sociodemographic Variables included	Yes		Yes		
Health Variables Included	No		Yes		
Interactions of Included Variables and pre-/post NLEA indicator	Yes		Yes		

Absolute t-ratios are in parentheses; ***=p<0.01, **=p<0.05, and *=p<0.10.

Table 7. Label Use Effects for Non-Hispanic White Women Estimated Across Various Time Periods

Period	Covariate unadjusted		Covariate adjusted	
	BMI (kg/m^2)	Obesity (%)	BMI (kg/m^2)	Obesity (%)
Pre-NLEA Years				
1991/1993	-0.35*	2.22*	0.10	0.39
	(1.81)	(1.67)	(0.58)	(0.30)
Pre/Post NLEA Years				
1991/1995	-0.43*	-1.38	-0.34	-1.14
	(1.65)	(0.78)	(1.39)	(0.61)
1993/1995	-0.77***	-3.60*	-0.45*	-1.50
	(2.77)	(1.90)	(1.65)	(0.77)
1991/1998	-0.28	-2.90**	-0.16	-2.81**
	(1.53)	(2.31)	(0.93)	(2.21)
1993/1998	-0.63***	-5.12***	-0.26	-3.20**
	(-2.98)	(3.55)	(1.28)	(2.23)

Absolute t-ratios are in parentheses; ***=p<0.01, **=p<0.05, and *=p<0.10.

Table 8. Differences and Difference-in-Differences in BMI Percentiles between1991/93 and 1995/98 by Label Use Status, for Non-Hispanic White Women

Percentile	Label Users			Label Non-User			
	1995/98	1991/93	Δ_u	1995/98	1991/93	Δ_n	$\hat{\delta}=\Delta_u-\Delta_n$
5	19.00	18.75	0.25	18.31	17.91	0.39	-0.14
15	20.56	20.19	0.37	20.09	19.53	0.56	-0.19
25	21.60	21.22	0.38	21.22	20.71	0.52	-0.14
35	22.61	22.21	0.40	22.46	21.66	0.80	-0.40
50	24.19	23.75	0.44	24.20	23.26	0.95	-0.50
65	26.47	25.72	0.75	26.55	24.99	1.56	-0.82
75	28.26	27.39	0.87	28.34	26.59	1.75	-0.88
85	30.68	29.93	0.74	31.07	29.17	1.90	-1.15
95	35.63	34.84	0.78	36.00	34.34	1.66	-0.88

Fig 1. Differences and Difference-in-Differences in BMI percentiles between pre- and post-NLEA periods by label use status for non-Hispanic white women

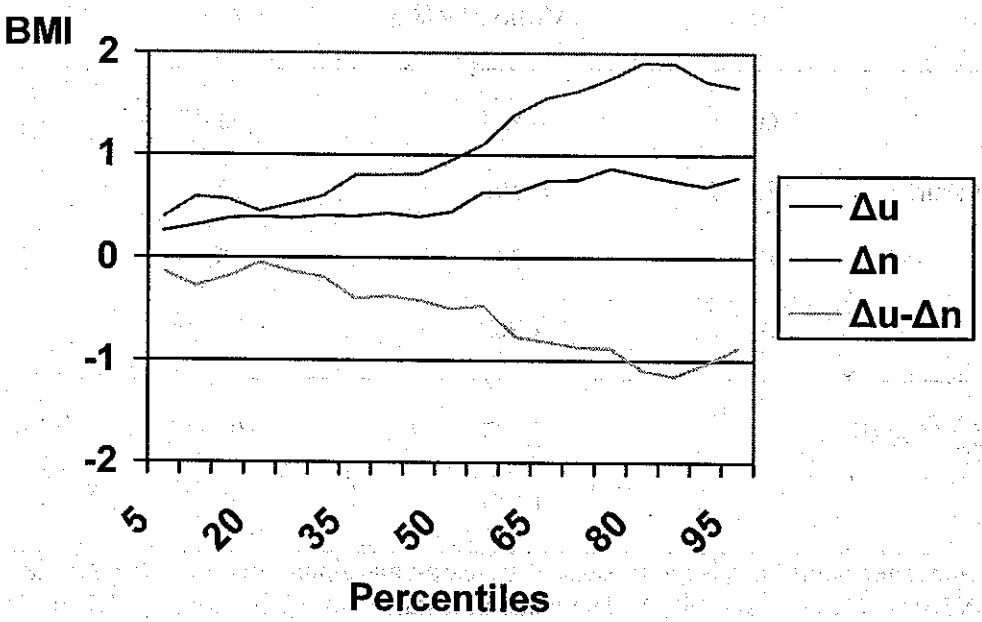

Table 9. Monetary Benefits from the NLEA

Source	Per-Person Value[a] ($)	Total Benefit (Million 1988 $)	Benefits over 20 years[b] (Million 1991 $)
Mortality	60	4,424	63,472
Medical Expenditures	35	1, 596	22,896
Absenteeism	13	579	8,308
Productivity	74	4,962	71,199
Total (Excluding Mortality)	-	7,137	102,402
Grand Total	-	11,561	165,875

[a]Derived from published sources. Mortality value is based on marginal reduction in death risk due to lower BMI from Calle et. al. (1999). The value of a statistical life is taken as $1.5 million (in 1988 $) as in Zarkin et al. (1993). Benefits from reduced medical expenditures and absenteeism due to lower BMI are from Finkelstein, Fiebelkorn, and Wang (2003, 2005). Benefits from higher productivity is from Cawley (2004).
[b]Discounted at 5 percent. The estimates are presented in 1988 and 1991 $ for comparison with FDA's estimates reported in Zarkin et al. (1993) and Food and Drug Administration (1993).

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DO NUTRITION LABELS IMPROVE DIETARY OUTCOMES?†

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SUMMARY

The disclosure of nutritional characteristics of most packaged foods became mandatory in the United States with the implementation of the Nutrition Labeling and Education Act (NLEA) in 1994. Under the NLEA regulations, a 'Nutrition Facts' panel displays information on nutrients such as calories, total and saturated fats, cholesterol, and sodium in a standardized format. By providing nutrition information in a credible, distinctive, and easy-to-read format, the new label was expected to help consumers choose healthier, more nutritious diets. This paper examines whether the disclosure of nutrition information through the mandatory labels impacted consumer diets. Assessing the dietary effects of labeling is problematic due to the confounding of the label effect with unobserved label user characteristics. This self-selection problem is addressed by exploiting the fact that the NLEA exempts away-from-home foods from mandatory labeling. Difference-in-differences models that account for zero away-from-home intakes suggest that the labels increase fiber and iron intakes of label users compared with label nonusers. In comparison, a model that does not account for self-selection implies significant label effects for all but two of the 13 nutrients that are listed on the label. Published in 2007 by John Wiley & Sons, Ltd.

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KEY WORDS: diet quality; difference-in-differences; health information; information regulation; mandatory disclosure

INTRODUCTION

Provision of health and nutrition information is an increasingly important strategy in promoting public health, particularly with regard to preventable risk factors associated with chronic disease, such as poor diet quality and obesity. In the United States, a landmark event in the dissemination of nutrition information to consumers was the enactment of the Nutrition Labeling and Education Act (NLEA) in 1990. The NLEA regulates nutrition labeling, health claims, and nutrient content claims for most foods sold in the United States. The US Food and Drug Administration (FDA) issued final regulations to implement the NLEA in January 1993, and these regulations took effect in May 1994. Under NLEA's authority, similar rules are enforced to regulate the voluntary labeling of raw fruits, vegetables and seafood by the FDA, and fresh meat and poultry by the US Department of Agriculture's Food Safety Inspection Service (FSIS). NLEA represented the first comprehensive overhaul of US food labeling laws in half a century. Prior to the NLEA's enactment, food labeling in the country was regulated by the Food, Drug, and Cosmetic Act of 1938 (Kurtzweil, 1993).

The cornerstone of labeling regulations promulgated by the FDA under the NLEA's mandate is the 'Nutrition Facts' panel (NFP), which nearly all packaged foods are required to carry. The panel provides information on serving size and servings per package or container, along with the per serving

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amounts and the percentage of daily values of nutrients such as calories, total and saturated fats, cholesterol, and sodium. For reference, the panel includes a table of recommended daily values of the nutrients for a 2000-calorie diet (US Food and Drug Administration, 1999). By providing nutrition information in a credible, distinctive, and an easy-to-read format, the new label was expected to help consumers choose healthier, more nutritious diets.

The purpose of this paper is to examine whether the disclosure of nutrition information through the mandatory NFP has had an impact on consumer diets. While NLEA's impact can be measured in many dimensions – the extent of label use and the comprehension of label information, for example – its influence on dietary intakes is of central policy interest. FDA's cost–benefit analysis for implementing the labeling regulations was based on predicted changes in consumer intakes of total fat, saturated fat, and cholesterol due to the new label information (Zarkin *et al.*, 1993). The rapid increase in obesity prevalence in the US has refocused attention on NLEA's impact and led the FDA to implement changes in the NFP format (US Food and Drug Administration, 2004). Besides measuring the impact of a landmark federal regulatory effort on an important intended outcome, estimating the dietary effects of nutrition labels will help in designing future policy initiatives, such as labeling for food eaten away from home.

Estimating the dietary impact of label use is challenging because label users and nonusers are typically not randomized in surveys that assess food intakes. Therefore, both label use and food intake are likely to be affected by factors that may remain unobservable to the researcher. This self-selection problem is addressed in the present study by a difference-in-differences (DD) identification strategy. Specifically, the effect of label use is estimated by exploiting the fact that while the NFP is mandatory for most foods sold in stores, restaurant foods and other food-away-from-home (FAFH) sources are exempt from the NFP requirement. As a result, label users – the treatment group – are exposed to the label (the NPP) in one setting but not in the other. Label nonusers in the same settings act as the control group. This quasi-experimental setup permits isolating the effect of label use by applying a DD estimator.

The next section provides some background to the study with an overview of the NLEA and previous research on NLEA's impact. The third section focuses on the empirical approach and presents the DD estimator and the identification strategy in greater detail. This is followed by a discussion of the data, dietary intake measures, and the explanatory variables used in the study. The fourth section presents the empirical results. The final section concludes with a discussion of the significance of the findings and potential limitations of the study.

BACKGROUND

NLEA's primary aim was to help consumers choose more healthful foods by providing them access to consistent, standardized, and credible nutrition information (Kurtzweil, 1993). The mandatory NFP was the major means for achieving this objective. Food producers and processors were allowed to display nutrition labels prior to NLEA under the voluntary labeling rules established by the FDA in 1975. However, the labels were required only if the packages displayed claims about the nutrient content of the food. Lack of standardization and relatively low coverage made it difficult for consumers to compare information across products and to interpret the information in a useful manner (Jessup, 2000). While only 60% of food products carried labels in 1990, 96% had labels in 1996 (Brecher *et al.*, 2000).

The impetus for the NLEA legislation was the growing evidence linking poor diets and obesity to chronic illnesses such as cardiovascular disease, diabetes and some types of cancer (Kurtzweil, 1993). The selection of nutrients listed on the NFP was based on these health concerns. The expectation was that consumers would use the nutrition information on the labels to make better dietary choices and thus achieve better health outcomes by avoiding illnesses associated with poor diets. As noted earlier,

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health improvements resulting from label-inspired dietary changes formed a significant part of FDA's cost–benefit analysis of the proposed labeling rules (Zarkin *et al.*, 1993).

Previous research has examined the several dimensions of NLEA's impact. Using data gathered eight months before and eight months after NLEA's implementation, Moorman (1996) found that the new labels helped consumers acquire and comprehend more nutrition information. Prevalence of potentially misleading health claims declined significantly following NLEA implementation (Mayer *et al.*, 1998). Other studies have specifically examined NLEA's impact on consumers' search for and use of nutrition information (Balasubramanian and Cole, 2002; Lin *et al.*, 2004) and on the degree of information unfolding in food markets (Mathios, 2000).

Relatively few studies, however, have evaluated NLEA's impact on dietary intakes. Some studies have examined correlations or regression-adjusted associations between label use and intakes and reported beneficial effects associated with label use (Kreuter *et al.*, 1997; Neuhouser *et al.*, 1999; Satia *et al.*, 2005; Weaver and Finke, 2003). These studies, which were based on self-reported label use, did not account for possible self-selection bias. Kim *et al.* (2000) also used self-reported label use to estimate the NLEA's dietary impact. However, they used a switching regression model to account for the endogeneity of label use and found significant effects for label use on the intakes of fats, cholesterol, sodium, and fiber. Their results relied on specific assumptions regarding the exclusion restrictions used to identify the label-use equation. The present study does not relay on exclusion restrictions for identifying the label effect. Instead, it uses label user–nonuser difference in a label-free or untreated situation to account for self-selection effects.

EMPIRICAL APPROACH

To estimate the dietary effects of the NFP, this study exploits a natural experiment provided by the NLEA's exemption of FAFH sources from the labeling requirement. As noted earlier, the NLEA requires nutrition labels for most foods sold in stores for at-home food preparation and consumption or for ready-to-eat (RTE) items consumed at home or elsewhere, while foods sold in restaurants, fast food places, and other away-from-home sources are exempt from labeling (Foulke, 1996). As a result, label users are exposed to the NFP information only for food-at-home (FAH) and not for FAFH, which constitutes a significant part of daily food intake. American consumers spend about 46% of their food budget on FAFH and obtain about 32% of their total daily caloric intake from FAFH (Guthrie *et al.*, 2002). A key provision in the NLEA regulations thus provides an exogenous source of variation for estimating the effect of the label on the label users.

To exploit this variation, suppose we have nutrient intakes of individuals separately from FAH and FAFH food sources. The total intake of the nutrient for an individual would be the sum of these parts. Since the NFP is available for at-home food only, the true measure of its impact should be assessed from at-home intake. This can be estimated from the following equation:

$$y_{ih} = \beta_{0h} + \beta_{1h}\text{LABEL}_i + \sum_{k=2}^{K} \beta_{kh} X_{ik} + \gamma_i + \epsilon_{ih} \tag{1}$$

where y_{ih} is the nutrient intake from FAH of the ith subject, LABEL_i is a binary indicator of label use, and X_{ki} denotes additional covariates. Equation (1) includes γ_i, which represents individual-level unobserved fixed effects; β_{0h}, β_{1h}, and β_{kh} are coefficients that capture observable effects specific to at-home intake.

The availability of intake from FAFH sources makes it possible to specify a similar equation

$$y_{ia} = \beta_{0a} + \beta_{1a}\text{LABEL}_i + \sum_{k=2}^{K} \beta_{ka} X_{ik} + \gamma_i + \epsilon_{ia} \tag{2}$$

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where y_{ia} is the intake of the same nutrient from FAFH, γ_i represents individual-level unobserved fixed effects that is common to both at-home and away-from-home intake and β_{0a}, β_{1a}, and β_{ka} are coefficients that capture effects specific to away-from-home intake.

Since labels are not available for away-from-home foods, β_{1a} gives the untreated effects specific to label users that are unrelated to the true label effect. This untreated effect and any individual-level fixed effects that may be correlated with label use are both potential sources of biases when estimating the label effect β_{1h} in Equation (1). These can be removed by subtracting Equation (2) from Equation (1). After rearranging the terms, the new equation can be written as

$$\Delta y_i = \delta_0 + \delta_1 \text{LABEL}_i + \sum_{k=2}^{K} \delta_k X_{ik} + \eta_i \tag{3}$$

where $\Delta y_i = y_{ih} - y_{ia}$, $\delta_0 = (\beta_{0h} - \beta_{0a})$, $\delta_1 = (\beta_{1h} - \beta_{1a})$, $\delta_k = (\beta_{kh} - \beta_{ka})$, and $\eta_i = (\epsilon_{ih} - \epsilon_{ia})$.

Note that Equation (3) is similar to a two-period fixed-effects panel model, except that the time invariant covariates are allowed to have time-varying effects. A similar DD model is discussed in Stock and Watson (2003) and employed in Card and Krueger (1994). In this study, allowing the effects of the fixed covariates to vary between FAH and FAFH as captured by coefficients β_{kh} and β_{ka} in Equations (1) and (2) or their difference δ_k in Equation (3) helps reduce any potential bias of the label effect due to the presence of effects that vary between FAH and FAFH and may be correlated with label use. Besides sociodemographic characteristics, we also include indicators for health status, time preference, and dietary attitudes to control for differential effects on FAH and FAFH.

If the covariate effects are assumed to be invariant across FAH and FAFH, then $\beta_{kh} = \beta_{ka}$, and therefore the δ_k terms fall off from Equation (3). In this case, the label effect δ_1 can be estimated from

$$\Delta y_i = \delta_0 + \delta_1 \text{LABEL}_i + \eta_i \tag{4}$$

or equivalently from

$$\delta_1 = (\bar{y}_h^1 - \bar{y}_h^0) - (\bar{y}_a^1 - \bar{y}_a^0) \tag{5}$$

where $\bar{y}$ denotes mean intake, 1 and 0 denote label use status, and h and a indicate intake from FAH and FAFH, respectively.

To estimate Equation (3), we require both FAH and FAFH intakes for each individual. However, most food consumption surveys measure intakes for only a few days (two days in the present case). With only a few days of intakes, a sizeable portion of the sample respondents may not report FAFH consumption, while nearly all would report at-home consumption. In the sample used in this study, the share reporting zero FAFH is 19.7% while the share reporting zero FAH is 0.5%. The zero away-from-home intakes for some individuals are handled by estimating Equation (3) using the Heckman selection procedure to account for the censoring. Strictly speaking, it is more efficient to estimate Equation (1) by ordinary least squares (OLS), Equation (2) by Heckman selection procedure, and then estimate the DD label effect as $\hat{\delta}_1 = (\hat{\beta}_{1h} - \hat{\beta}_{1a})$. In practice, such estimates were similar to estimates from Equation (3) and these are the ones reported here.

DATA AND VARIABLES

The data for this study were obtained from the US Department of Agriculture's 1994–1996 Continuing Survey of Food Intakes by Individuals (CSFII) and the companion Diet and Health Knowledge Survey (DHKS). The CSFII was based on a nationally representative sample of noninstitutionalized persons residing in the United States. In the 1994–1996 CSFII, food intake data for selected persons from a screened sample of 9664 households were collected through in-person interviews on two nonconsecutive

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days, using the 24-h dietary recall method. Detailed sociodemographic information of the households and individuals was also gathered. By combining the foods reportedly eaten with a nutrient composition database, CSFII public-use files provide information on the survey respondents' intakes of various macronutrients, vitamins, and minerals. The DHKS was a telephone follow-up to the CSFII. One individual aged 20 or above who met the eligibility criteria was selected at random from each household. DHKS gathered responses to questions on nutrition knowledge, attitudes, and dietary habits of the respondents, as well as on their use of nutrition labels. Since the DHKS respondents also provided their food intakes in the CSFII, their label-use habit and dietary intakes can be linked. The 1994–1996 CSFII–DHKS sample had a response rate of 74%. Public-use files provide detailed documentation of these surveys (US Department of Agriculture, 1998).

For each item of food consumed by a respondent, CSFII provides information on where the item was obtained. This permits an individual's food and nutrient intakes to be broken down by source. To link label use to the intake, at-home food is defined as food purchased at a store, such as a grocery store, a convenience store, or a supermarket. Almost all packaged foods sold in these venues carry the NFP. Labeling of raw meat and poultry products is similar to packaged products, except that labeling is voluntary. In 1996, 58% of raw meat and poultry products sold in supermarkets carried labels, according to the FSIS. Therefore, some of the at-home meat purchases may have been without labels. We could not exclude such purchases since there is no clear-cut indicator of labeled and nonlabeled meats. Fresh fruits and vegetables also fall under the voluntary labeling rule and label information is not likely to be available when customers choose the quantities. We report results after excluding fresh foods and vegetables to check the sensitivity of results to the inclusion of some nonlabeled foods among the at-home foods.

Away-from-home food is defined as that purchased from all other sources that are exempt from NLEA-mandated NFP labeling requirement, such as a restaurant or cafeteria. Although these venues increasingly provide nutrition information in some format or other, none of them have an NFP-type disclosure and any type of disclosure was likely rare during 1994–1996 (Variyam, 2005).

Equation (3) was estimated for all nutrients that are *required* to be listed on the NFP. These are total calories (energy), total fat, saturated fat, cholesterol, fiber, protein, carbohydrate, sugars, sodium, calcium, iron, vitamin A, and vitamin C (US Food and Drug Administration, 1999). A direct measure of FDA-defined 'sugars' is not available in the CSFII. Instead, intake of 'added sugars' provided by the CSFII is used, which is based on the Food Guide Pyramid definition of sugars (see Guthrie and Morton, 2000 for details). The initial study sample included 5649 CSFII–DHKS individuals who provided complete intake data for two days. After excluding respondents with incomplete information for the explanatory variables, the final sample size was 5439. Out of this sample, 4338 individuals reported both at-home and away-from-home intakes over the two days.

The CSFII–DHKS surveys were based on a stratified, multistage area probability sampling design. For analytical purpose, combined three-year sampling weights and design information are provided with the public-use data sets. These were used in the empirical analysis to obtain weighted estimates and standard errors that are adjusted for stratification and clustering. All analyses in this study were conducted using the survey procedures in STATA.

Explanatory variables

Table I reports the explanatory variables and their means. The main variable of interest, the label-use indicator $LABEL_i$, was obtained from responses to the DHKS question: 'Now think about food labels. When you buy foods, do you use the nutrition panel that tells the amount of calories, protein, fat, and such in a serving of the food often, sometimes, rarely, or never?' The original scale was dichotomized so that Always/Sometimes = 1 and Rarely/Never/Never Seen = 0. Sixty-six percent of adults report using the label during the 1994–1996 period. This is similar to FDA surveys, which

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Table I. Explanatory variables and weighted means

Variable	Mean
Region (northeast omitted)	
Midwest	0.23
South	0.35
West	0.22
Urbanization (city omitted)	
Suburb	0.46
Nonmetro	0.21
IPR: income-poverty ratio (<131% omitted)	
131–350%	0.41
>350%	0.43
Education (years)	13.1
Age (years)	45.2
Female	0.52
Race/ethnicity (non-Hispanic white omitted)	
Non-Hispanic black	0.11
Non-Hispanic other	0.04
Hispanic	0.09
Household WIC recipient	0.04
Household food stamps recipient	0.08
On any special diet	0.17
Year (1994 omitted)	
1995	0.33
1996	0.33
LABEL	0.66
Health (excellent/very good/good)	0.87
Smoker	0.25
Born to be fat or thin (strongly/somewhat disagree)	0.40
Diet–health link (strongly/somewhat disagree)	0.91
No reason to change diet (strongly/somewhat disagree)	0.52
Retired	0.16
Ratio of full-time employed adults to household size	0.38
N[a]	5439

[a] The sample size available for regression models. The sample sizes used to estimate the means of explanatory variables reported here may vary slightly.

indicate label use at 70 and 69% in 1994 and 1995, respectively (US Food and Drug Administration, 2004).

Several characteristics of individuals and their households were included as covariates to control for differences in intakes between FAH and FAFH. These were age, the square of age, gender, race/ethnicity, years of education, household income expressed as percent of poverty threshold (which adjusts for household size and annual inflation rate), region, urbanization, whether anyone in the household receives Women, Infants, and Children (WIC) or food stamp program benefits, whether the individual was on any special diet, and indicators for the survey year.

While these explanatory variables control for key sociodemographic differences, health status, time preference, and dietary attitudes that may be correlated with label use may impact FAH and FAFH intakes. To control such effects, we include several additional explanatory variables. These are dummy variables for self-reported health status, smoking status, and three dietary attitudes. The attitude variables include those indicating self-efficacy ('Some people are born to be fat and some thin; there is

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not much you can do to change this'), perceived diet–disease link ('What you eat can make a big difference in your chance of getting a disease, like heart disease or cancer') and intention for dietary change ('The things I eat and drink now are healthy so there is no reason for me to make changes').

Table I also includes two variables that we use to identify the Heckman selection equations to account for individuals who report zero FAH or FAFH intakes. These are the ratio of the number of full-time employed adults to the household size and a dummy variable indicating whether the individual is retired. We tried other variables including the number of children aged 5–12, employment hours, and weekend dummy variables. While their effects were less consistent across selection equations for all nutrients, the estimated label effects results were similar.

The 1994–1996 CSFII–DHKS was designed to be representative of the US population. The combined three-year sampling weights provided with the survey data were created so as to make target variables in the weighted samples attain standard US population estimates for 1994–1996 (US Department of Agriculture, CSFII documentation, Tables 5–7 through 5–10). For example, the population targets for adult (age 20+) food stamps participation are 6.2% for males and 9.7% for females. Our estimates for the combined sample is roughly in the middle, 8%. For Hispanics, the targets are 9.4 and 8.6%; our estimate is 9%. For IPR < 131% the targets are 12.5 and 18.6%; our estimate is 16%. Thus, our weighted sample is very close to the population levels targeted by the CSFII.

ESTIMATION AND RESULTS

The absolute amounts of food and nutrient intakes are higher at home than away from home. To treat at-home and away-from-home intakes on an equal basis, all intakes were expressed in relative terms. Energy intake was expressed as calories per kilogram of food (that is, as energy density). All other nutrients were expressed as quantities per 1000 calories of energy (nutrient density).

Table II reports simple DD estimates from Equation (5). The first set of columns give the mean intakes of nutrients from FAH for label users and nonusers, and the difference between the means. Label users have significantly more healthful intakes than nonusers of all nutrients except energy and sodium. For these two nutrients, the differences are statistically insignificant. (Whether the

Table II. Difference-in-differences estimates of the effect of nutrition label use on nutrient intakes (regression unadjusted), 1994–1996

	Food at home			Food away from home			
Nutrient	Label user	Nonuser	Difference	Label user	Nonuser	Difference	Difference-in differences
Energy (calories)	879.2	892.4	−13.2	1204.3	1249.4	−45.1*	31.9
Total fat (g)	32.5	36.0	−3.6***	39.6	41.4	−1.8***	−1.8**
Saturated fat (g)	10.9	12.5	−1.6***	12.8	13.8	−1.1***	−0.5
Cholesterol (mg)	117.2	136.8	−19.6***	135.8	150.6	−14.9**	−4.8
Fiber (g)	9.1	7.5	1.6***	8.1	7.6	0.6*	1.1***
Protein (g)	38.4	36.5	1.9***	40.5	40.3	0.2	1.7*
Carbohydrate (g)	139.0	130.1	8.9***	118.3	113.4	4.9***	4.0*
Added sugars (tsp)	9.6	11.1	−1.6***	9.1	9.7	−0.6	−0.9
Sodium (mg)	1655.9	1646.6	9.2	1817.7	1875.0	−57.4	66.6
Calcium (mg)	433.7	382.9	50.8***	362.0	339.4	22.6**	28.1
Iron (mg)	8.9	7.6	1.3***	7.3	6.9	0.4***	0.9***
Vitamin A (μg RE)[a]	621.6	497.3	124.3***	554.3	435.5	118.7*	5.6
Vitamin C (mg)	65.6	50.8	14.8***	51.1	40.4	10.8***	4.0

Note: Energy is expressed in calories per kg of food. All other nutrients are expressed in quantity per 1000 calories.
***$p<0.01$, **$p<0.05$, and *$p<0.1$.
[a] Micrograms retinol equivalents.

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higher carbohydrate intake of label users compared with nonusers is more healthful could be disputed. For the present purposes, a higher carbohydrate intake is taken as nutritionally beneficial.) While these estimates clearly show that label users consume better quality diets at home than label nonusers, the benefit cannot be attributed fully to their use of the NFP to purchase at-home foods (the treatment effect). The reason is evident in the second set of columns, which reports the mean nutrient intakes of label users and nonusers from FAFH, where the NFP information is unavailable. Here too label users have more healthful intakes than nonusers, with significantly lower intakes of energy, fats, and cholesterol and higher intakes of fiber, carbohydrate, calcium, iron, and vitamins A and C.

The FAFH differences capture the tendency of label users to consume better diets than label nonusers, independent of label availability. In the final column of Table II, this effect is subtracted out from the at-home difference between label users and nonusers, to give the DD estimate of the label effect. The net result is that label users still have more healthful diets than nonusers, but to a lesser degree than was evident from the FAH differences. For example, label users on average consumed 1.6 g less saturated fat per 1000 calories than label nonusers from at-home foods. However, label users also consumed 1 g less saturated fat per 1000 calories than label nonusers FAFH foods. The net label effect of a half-gram less saturated fat per 1000 calories is statistically insignificant. A positive label effect on at-home vitamin A intake also becomes insignificant when the label effect on away-from-home intake is subtracted out. Significant net label effects are obtained for total fat, fiber, protein, carbohydrate, and iron.

Regression results

Although Equation (5) removes individual fixed effects, some effects may have differential impact on FAH and FAFH. Also, Equation (5) was estimated without accounting for the censoring due to zero FAFH intakes reported over the 2 days of dietary recall. These issues are addressed with the estimation of Equation (3). In Table III, models 2 and 3 report regression-adjusted DD estimates of the label effect, $\hat{\delta}_1$, from two specifications of Equation (3). Model 2 adjusts for several sociodemographic characteristics and special diet status, and model 3 includes additional controls for health status, smoking, and dietary attitudes. Both models are estimated by the maximum likelihood Heckman procedure, adjusted for sampling weights and survey design. The full set of coefficient estimates for model 2 are reported in Appendix Table AI. The coefficient estimates for the selection equation for the nutrients were very similar. The estimates for iron intake are reported in Appendix Table AII.

As the inverse Mills ratio (lambda) coefficients reported in Table AI indicate, there is significant selection effects due to greater probability of reporting FAFH intakes for some but not all nutrients. Table AII estimates suggest that household income, education level of the individual, residence in the midwest, and the ratio of adult employed full time to the household size are positively related with the probability of eating away from home, while age, being a female, Hispanic ethnicity, and non-Hispanic other ethnicity are negatively related. In Table AI, several sociodemographic variables, including income, education, age, race/ethnicity, and urbanization are statistically significant, indicating their differential effect on FAH intakes compared with FAFH intakes. This indicates the importance of allowing the effects of these observables to vary between FAH and FAFH.

Turning to the main estimates of interest, the LABEL coefficient estimates in Table III, model 2 suggest that the use of the NFP has a nutritionally beneficial effect on the intakes of fiber and iron. The estimates imply that the at-home fiber and iron intakes of label users increase by 0.69 g and 0.65 mg, respectively, per 1000 calories due to the NFP availability – about 7% increase in consumption from the mean intake level in both cases. Comparing model 2 results with the label effects reported in final column of Table II, it can be seen that after adjustment for sociodemographic differences, label effects for total fat, protein, and carbohydrate are no longer statistically significant.

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Table III. Regression estimates of the effect of nutrition label use on nutrient intakes

Nutrient	Model 1: OLS FAH only[a]		Model 2: ML Heckman-corrected DD regression[b]		Model 3: ML Heckman-corrected DD regression with additional controls[c]	
Energy	18.5	(15.7)	32.3	(30.5)	22.06	(33.3)
Total fat	−2.26***	(0.46)	−0.88	(0.73)	−0.61	(0.79)
Saturated fat	−1.08***	(0.18)	−0.25	(0.32)	−0.16	(0.34)
Cholesterol	−9.15*	(5.34)	−0.97	(8.27)	−2.65	(7.56)
Fiber	1.03***	(0.16)	0.69**	(0.33)	0.74**	(0.32)
Protein	1.82***	(0.63)	0.79	(0.94)	0.47	(0.89)
Carbohydrate	6.45***	(1.32)	2.21	(2.02)	2.14	(2.10)
Added sugars	−1.07***	(0.37)	−0.82	(0.54)	−0.89	(0.57)
Sodium	4.66	(36.3)	−15.5	(97.1)	−6.58	(95.94)
Calcium	34.0***	(10.9)	21.9	(18.8)	15.86	(19.60)
Iron	0.98***	(0.15)	0.65***	(0.21)	0.63***	(0.23)
Vitamin A	63.2**	(28.5)	24.9	(59.5)	30.8	(58.4)
Vitamin C	7.34**	(3.09)	0.24	(7.29)	0.25	(7.67)

Note: Standard errors in parentheses; ***$p<0.01$, **$p<0.05$, and *$p<0.10$. Only the coefficient for the LABEL variable is reported. All models include indicators for region, urbanization, income–poverty ratio, sex, household WIC participation, household food stamps participation, and whether the individual is on any special diet, as well as years of educational attainment, age and age squared as additional regressors; see Table I for definitions of these variables.

[a] Estimates from Equation (1) estimated by OLS regression. The dependent variable is the intake of the nutrient from FAH.

[b] Estimates from Equation (3) estimated by maximum likelihood Heckman selection procedure to account for zero intakes. The dependent variable is difference in the intake of the nutrient between FAH and FAFH. The selection equation includes indicators for region, urbanization, income–poverty ratio, sex, household WIC participation, household food stamp participation, whether the individual is on any special diet, years of educational attainment, age, ratio of adults employed full time to the household size, and whether the individual is retired.

[c] The model also includes dummy variables for self-reported health, smoking, and three dietary attitude measures as additional regressors.

Model 3 results in Table III suggest that the addition of health variables and dietary attitudes does not alter the conclusions from model 2. These additional variables were chosen because they are likely to be correlated with unobservable variables that may influence dietary choices of FAH, FAFH, and label use. For example, since smoking has been shown to increase cancer and heart disease risk, a choice to smoke is related to time preference for health. Time preference could also affect food choice at home and away from home, as well as label use. To the extent that the additional variables in model 3 control for the differential effects of unobservables on FAH and FAFH, the results show that the positive label effects for fiber and iron are unlikely to be the result of correlation between the unobservables and label use.

To put the DD results into perspective, model 1 in Table III reports label effects from Equation (1) estimated using OLS. The explanatory variables included are the same set of sociodemographic characteristics and special diet status used in model 2. The LABEL coefficients in model 1 give the difference in the FAH intakes of label users compared with label nonusers. Neither the untreated differences nor the unobserved individual effects are removed in Equation (1). As can be seen, compared with label nonusers, label users are implied to have significantly different intakes of all but energy and sodium. In contrast, once the untreated differences and individual fixed effects are removed in Equation (3), the only statistically significant effects are for fiber and iron.

As noted before, the presence of unobserved factors that are correlated with label use and that differentially affect FAH and FAFH may bias the estimated label effect. While the Heckman selection procedure for zero FAFH intakes as well as the use of additional controls in Model 3 suggests that such bias may be limited, it cannot be ruled out. An additional check for bias can be conducted based on the fact that labels are very prominent and universal on some of foods whereas labels are scarce for some other foods because labeling is voluntary. Specifically, the NFPs are displayed on nearly all breakfast cereal packages and this sector has played a large role in product promotion using label information. In

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Table IV. Regression estimates of the effect of nutrition label use on nutrient intakes: sensitivity of model 2 results

Nutrient	Label use for breakfast cereals	Label use for fresh fruits and vegetables	Excluding fresh fruits and vegetables
Fiber	1.17*** (0.39)	−0.18 (0.45)	0.68* (0.35)
Iron	0.89*** (0.31)	−0.66 (0.40)	0.75*** (0.22)

Note: Standard errors are given in parentheses; ***$p<0.01$, **$p<0.05$, and *$p<0.10$.
Only the coefficient estimate for the LABEL variable is reported. Column 1 re-estimates model 2 in Table III except that the LABEL variable indicates whether the respondent uses food label information when buying breakfast cereals. In column 2, the LABEL variable indicates whether the respondent uses food label information when buying fruits and vegetables. In column 3, the LABEL variable is same as in model 2 in Table III. However, the dependent variable (fiber or iron intake) is calculated after excluding fresh fruits and vegetables from the intake data.

contrast, for fresh fruits and vegetables, labeling is voluntary and not available when purchased in random weight (quantity chosen and packed by customer), although some information may be available from store displays or brochures. Unobservable factors, if present, should operate similarly on different types of label use. If this is the case, then the presence of unobserved factors would be indicated by similar effects for label use for breakfast cereals and for fruits and vegetables on dietary outcomes. Conversely, if information difference between labels for breakfast cereals and fresh fruits and vegetables is driving the label effects, then the use of labels for breakfast cereals is likely to have a greater impact on nutrient intakes compared with the use of labels for fresh fruits and vegetables. The first two columns in Table IV report the results from this sensitivity test. The food-specific label use variables are developed from the question 'When you buy (food product), do you look for nutrition information on the food label often, sometimes, rarely, or never?' The label use for fresh fruits and vegetables has no impact on fiber and iron intake whereas label use for breakfast cereals has a significant impact. This suggests that the estimated label effect is related to the information content and not due to the effect of unobserved factors.

Since labeling may not be available for some of the at-home foods such as fresh fruits and vegetables, FAH nutrient intakes may not completely reflect intakes from labeled foods. Column 3 of Table IV reports estimated label effects after excluding fresh fruits and vegetables from the computation of FAH intakes. The estimates are very similar to model 2 estimates in Table III suggesting that the inclusion of partially labeled foods does not alter the results.

CONCLUSIONS

Using a quasi-experimental approach to control for unobserved selection effects, this study presents evidence that the Nutrition Facts panel mandated by the NLEA had a modest but beneficial impact on dietary intakes of Americans. Those who reported using the NFP when buying food had significantly higher fiber and iron intakes compared with those who rarely or never used the NFP. These nutritional effects have public health significance because fiber is underconsumed by large proportions of adult Americans and increased iron intake is beneficial for many segments of the population, particularly premenopausal women. Why does label use increase the intakes of fiber and iron? A plausible explanation is that the increase may reflect the influence of nutrition labels on the choice and consumption of ready-to-eat (RTE) breakfast cereals. RTE cereals are the top source of iron in the diets of adult Americans, contributing 17% of their iron intake (Cotton *et al.*, 2004). For fiber, RTE cereals are the fourth major source, contributing 9% of total fiber intake. Previously, Ippolito and Mathios (1990) have shown that the relaxation of health claim regulations in the breakfast cereal market in the 1980s led to a change in consumer behavior associated with fiber consumption.

After accounting for key sources of biases, the study finds no evidence that label use is associated with a reduction in total fat, saturated fat, or cholesterol intake. This is different from projected changes in

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these nutrients used in FDA's regulatory impact analysis of labeling rules (Zarkin *et al.*, 1993) and previous findings on the impact of label use on these nutrients (Kim *et al.*, 2000; Kreuter *et al.*, 1997; Neuhouser *et al.*, 1999). Our results suggests that caution is needed in using decreased intakes of these nutrients as sources of benefits (through reductions in heart disease and cancer) for evaluating future nutrition labeling policies such as labeling for away-from-home foods.

The study has some limitations. The estimated label effects do not account for potential benefits of label use gained by substitutions among nutrients. Consumers may act as if they have nutrient or health risk budgets and the label information may allow them to substitute foods, with little or no net change in nutrient intakes. Another limitation is that this study only estimates the average label effect over all label users. The label effects, however, may vary across sociodemographic groups and such information could be potentially important in assessing the distributional impact of the labeling regulation.

APPENDIX

Models 1 and 2 are estimated by the maximum likelihood Heckman procedure, adjusted for sampling weights and survey design. The full set of coefficient estimates for model 2 are reported in Table AI. The estimates for iron intake are reported in Table AII.

Table AI. Coefficient estimates from ML Heckman-corrected difference-in-difference regressions (Model 2)

Variables	Energy	Total fat	Saturated fat	Cholesterol	Fiber	Protein	Carbohydrate
Part 1							
Intercept	41.43	−4.04	−0.59	96.77*	−2.86	−6.78	25.32*
	(181.88)	(4.89)	(1.53)	(51.31)	(1.79)	(5.73)	(13.32)
Midwest	−10.49	−0.99	−0.37	10.67	−0.71	1.47	−1.24
	(68.43)	(1.30)	(0.56)	(10.32)	(0.50)	(1.30)	(3.24)
South	10.82	−2.00*	−0.41	9.16	−0.77	−1.89	3.41
	(58.89)	(1.17)	(0.59)	(10.17)	(0.49)	(1.51)	(3.15)
West	−5.95	−0.91	−0.38	−1.89	0.13	−0.07	−0.66
	(55.68)	(1.26)	(0.53)	(11.81)	(0.58)	(1.45)	(2.77)
Suburb	−22.80	−0.32	0.43	−0.65	−0.69*	−0.13	−0.89
	(58.48)	(0.90)	(0.36)	(7.89)	(0.40)	(1.20)	(2.86)
Nonmetro	−40.13	0.40	0.58	−21.22**	−1.24**	−3.14**	−0.30
	(61.32)	(1.10)	(0.42)	(8.30)	(0.52)	(1.25)	(2.90)
IPR 131–350%	−42.24	1.60	0.35	−23.45*	0.42	−0.40	−4.64
	(63.71)	(1.42)	(0.77)	(13.49)	(0.47)	(1.91)	(3.28)
IPR > 350%	−109.29	−0.39	−0.12	−48.57**	0.51	−1.99	1.87
	(71.25)	(1.54)	(0.81)	(17.48)	(0.62)	(2.13)	(4.18)
Education	−10.37	−0.48**	−0.11	−4.29**	0.08	−0.36*	1.42**
	(7.28)	(0.19)	(0.08)	(2.00)	(0.06)	(0.21)	(0.57)
Age	−1.11	0.24	0.02	−0.43	0.13**	0.46**	−1.13**
	(5.01)	(0.16)	(0.06)	(1.11)	(0.06)	(0.20)	(0.47)
Age squared ($\times 10^{-2}$)	0.04	−0.00	−0.00	0.01	−0.00**	−0.44**	0.94*
	(0.05)	(0.00)	(0.00)	(0.01)	(0.00)	(0.21)	(0.47)
Female	−31.62	−1.13	−0.04	8.88	−0.24	1.21	1.86
	(45.94)	(0.70)	(0.33)	(8.03)	(0.36)	(0.91)	(1.96)
Non-Hispanic black	−88.29	0.38	0.09	34.63***	−0.97	−0.38	−3.46
	(66.49)	(1.22)	(0.68)	(12.46)	(0.61)	(1.64)	(4.05)
Non-Hispanic other	447.22***	−0.44	0.21	84.56***	−1.05	3.49	−3.67
	(100.41)	(2.25)	(0.93)	(21.39)	(0.71)	(3.00)	(7.52)
Hispanic	192.48***	−0.26	−0.13	71.29***	0.46	0.40	−1.41
	(57.99)	(1.45)	(0.64)	(17.27)	(0.53)	(1.74)	(4.68)
WIC	40.88	2.17	0.60	1.23	1.43**	−3.62	−3.31
	(72.01)	(2.00)	(0.97)	(22.36)	(0.68)	(4.50)	(7.94)
Food stamps	−134.84*	0.30	−0.00	−18.75	−0.68	−7.15**	3.21
	(59.20)	(1.79)	(1.12)	(12.42)	(0.78)	(3.04)	(4.82)

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Table AI. *Continued*

Variables	Energy	Total fat	Saturated fat	Cholesterol	Fiber	Protein	Carbohydrate
Special diet	38.18	−1.49	−0.67	−2.90	1.05**	1.36	3.42
	(57.77)	(0.97)	(0.43)	(11.41)	(0.44)	(1.30)	(2.54)
1995	32.13	−0.45	−0.60	−7.61	0.34	−0.78	3.81
	(43.72)	(1.04)	(0.49)	(8.62)	(0.43)	(1.17)	(2.96)
1996	95.64**	0.33	−0.59	11.20	0.98*	−0.52	1.74
	(37.92)	(0.79)	(0.40)	(8.95)	(0.49)	(1.03)	(2.65)
LABEL	32.35	−0.88	−0.24	−0.97	0.69**	0.79	2.21
	(30.49)	(0.73)	(0.32)	(8.27)	(0.33)	(0.94)	(2.02)
F-value	2.89***	1.36	0.67	4.39***	4.42***	2.14**	1.90*
Lambda	−828.90***	−0.88	0.08	−149.57***	0.14	0.20	2.66
	(42.78)	(1.33)	(0.31)	(28.60)	(0.20)	(0.84)	(2.04)

Variables	Added sugars	Sodium	Calcium	Iron	Vitamin A	Vitamin C
Part 2						
Intercept	4.17	845.91*	56.43	4.81***	674.98**	117.66***
	(5.64)	(433.37)	(89.70)	(0.50)	(312.41)	(32.24)
Midwest	0.32	−219.23	36.52**	0.38	−131.00	−9.14
	(1.08)	(198.25)	(15.17)	(0.52)	(120.59)	(6.94)
South	1.95**	−36.10	7.90	−0.06	−93.55	−10.78
	(0.78)	(89.71)	(17.51)	(0.42)	(72.42)	(7.35)
West	−0.47	−142.84	28.53	0.38	5.28	−3.91
	(0.78)	(114.16)	(22.55)	(0.56)	(57.99)	(8.53)
Suburb	0.92	71.71	8.43	−0.71**	−112.85	−1.46
	(0.70)	(88.38)	(21.67)	(0.33)	(49.69)	(8.69)
Nonmetro	1.46*	−162.38	−0.59	−1.08***	−190.57	−32.01***
	(0.80)	(197.89)	(22.59)	(0.32)	(109.07)	(11.22)
IPR 131–350%	−0.35	−347.22*	39.11	0.01	−44.48	24.22*
	(1.73)	(173.99)	(25.30)	(0.39)	(59.07)	(14.14)
IPR > 350%	0.37	−809.19**	43.45*	0.32	48.76	−32.81**
	(2.74)	(345.89)	(25.87)	(0.40)	(63.91)	(13.71)
Education	0.18	−24.31	3.34	0.05	−11.41	0.75
	(0.19)	(16.60)	(3.80)	(0.05)	(10.38)	(1.16)
Age	−0.25**	5.58	−5.57**	−0.10**	−24.14**	−2.67***
	(0.12)	(10.38)	(2.69)	(0.04)	(9.12)	(0.92)
Age-squared ($\times 10^{-2}$)	0.19*	0.85	5.99**	0.10**	17.44*	3.04***
	(0.11)	(9.74)	(2.57)	(0.04)	(9.14)	(0.90)
Female	0.36	41.62	5.34	0.20	43.74	8.79
	(0.62)	(105.48)	(17.38)	(0.22)	(82.79)	(7.98)
Non-Hispanic black	0.12	39.45	−35.98	−1.23***	−158.26	−5.34
	(1.13)	(140.52)	(40.67)	(0.39)	(96.45)	(16.32)
Non-Hispanic other	−2.34	508.44*	−22.54	−0.71	−91.51	61.15**
	(2.82)	(260.43)	(57.05)	(0.76)	(102.40)	(23.72)
Hispanic	−1.71	575.79**	−24.51	−1.02**	−100.06	50.85***
	(2.49)	(241.69)	(30.51)	(0.42)	(97.20)	(15.94)
WIC	−1.71	−109.99	−55.98	−0.34	−111.12	7.54
	(2.49)	(153.05)	(45.49)	(0.71)	(96.92)	(8.34)
Food stamps	2.28*	11.91	10.35	−0.98*	92.36	−18.80
	(1.25)	(117.19)	(50.02)	(0.56)	(115.75)	(15.59)
Special diet	−0.41	237.23*	27.38	0.46	−142.22	3.99
	(0.86)	(139.61)	(24.08)	(0.40)	(128.06)	(8.66)
1995	0.16	194.69	−2.08	0.03	149.29*	11.11
	(0.76)	(158.05)	(21.95)	(0.33)	(84.34)	(7.53)
1996	−0.60	245.97	−9.11	0.34	180.86*	9.54
	(0.84)	(173.01)	(18.95)	(0.29)	(99.90)	(6.92)
LABEL	−0.82	−15.51	21.86	0.65***	24.85	0.24
	(0.54)	(97.06)	(18.82)	(0.21)	(59.46)	(7.29)
F-value	2.45**	4.89***	2.12	3.30***	2.65**	3.74***
Lambda	−0.02	−2307.17***	7.73	0.41*	1.43	−147.61***
	(9.78)	(778.57)	(7.94)	(0.21)	(52.06)	(24.43)

Note: Standard errors are given in parentheses; $N = 5439$; ***$p<0.01$, **$p<0.05$, and *$p<0.1$.

Table AII. Probit equation for Heckman-correction of iron intake

Variable	Coefficient estimate
Intercept	0.68***
	(0.22)
Midwest	0.16*
	(0.09)
South	−0.03
	(0.07)
West	0.02
	(0.10)
Suburb	−0.04
	(0.06)
Nonmetro	0.11
	(0.09)
IPR 131–350%	0.24***
	(0.06)
IPR > 350%	0.46***
	(0.08)
Education	0.03***
	(0.01)
Age	−0.01***
	(0.00)
Female	−0.10**
	(0.05)
Non-Hispanic black	0.00
	(0.10)
Non-Hispanic other	−0.64***
	(0.13)
Hispanic	−0.43***
	(0.11)
Special diet	−0.02
	(0.07)
1995	−0.05
	(0.07)
1996	−0.16***
	(0.05)
Retired	−0.05
	(0.07)
Full-time employed adults	0.34***
	(0.10)

Note: Standard errors are given in parentheses; ***$p<0.01$, **$p<0.05$, and *$p<0.1$.

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Health Econ. **17**: 695–708 (2008)
DOI: 10.1002/hec

UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

NEW YORK STATE RESTAURANT ASSOCIATION,

Plaintiff,

-against-

NEW YORK CITY BOARD OF HEALTH, NEW YORK CITY DEPARTMENT OF HEALTH AND MENTAL HYGIENE, and Thomas R. Frieden, In His Official Capacity as Commissioner of the New York City Department of Health and Mental Hygiene,

Defendants.

No. 2008 Civ. 1000 (RJH)

DECLARATION OF KENT A. YALOWITZ

Kent A. Yalowitz declares as follows:

1. I am a member of the bar of this Court and of the firm of Arnold & Porter LLP, counsel for the Plaintiff New York State Restaurant Association ("NYSRA") in the above-captioned case.

2. This declaration is based on documents which we received within the last few days from the Defendants in response to a FOIL request. These documents are submitted with this declaration in order to correct possible misimpressions regarding the status of a study conducted by the New York City Department of Health and Mental Hygiene ("DOHMH") upon which defendants rely to support their contention that New York City Health Code section 81.50 ("Regulation 81.50") will reduce the prevalence of obesity among New York City residents.

3. The study, a survey of calorie information and calorie ordering at New York City chain restaurants conducted in 2007, was heavily relied on by the DOHMH as empirical evidence that Regulation 81.50 will have its intended effect. Notice of Adoption of Regulation 81.50, Jan. 22, 2008, at 4, 6-7. Critiques of the study and of its interpretation by DOHMH were

presented in the Jan. 29, 2008 Declaration of David B. Allison, Ph.D., submitted by plaintiff in support of its motion for declaratory and injunctive relief. In response, defendants submitted Declarations by DOHMH Commissioner Frieden and Deputy Commissioner Mary T. Bassett, co-authors of the study, Frieden Declaration 2/8/08 ¶¶ 32, 43; Bassett Declaration 2/8/08 ¶¶ 3-11. Deputy Commissioner Bassett reported that the DOHMH study was "currently under review by a peer-reviewed journal." She failed to disclose, however, that the study has been rejected by two journals, the reasons given for rejection, and her subsequent personal involvement in recruiting peer reviewers to review a study of which she was the lead author. This information has recently come to light as a result of DOHMH's belated response to a Freedom of Information Law request for its records on the study. Those records establish the facts set forth below. Copies of the relevant records are attached.[1]

4. The DOHMH first attempted to have the study published in Morbidity and Mortality Weekly Reports ("MMWR"), a weekly publication of the United States Centers for Disease Control. In November 2007, Commissioner Frieden wrote to MMWR Editor Dr. Frederic Shaw asking to have the study published. Dr. Shaw requested an abstract of the study, which Commissioner Frieden provided. On December 29, 2007, Dr. Shaw wrote back, rejecting the article with the following comments:

> "I read the article carefully and asked a few others to also take a look, and I do not feel it bears publishing in MMWR as it stands. The conclusions being drawn by the study are, of course,

[1] On November 15, 2007, we made a Freedom of Information Law ("FOIL") request for all records within the custody or control of the DOHMH that relate to the survey. The DOHMH "granted" it in full and produced certain records. See Milburn letters of 10/24/07 and 12/28/07 and DOHMH letters of 12/6/07 and 1/8/08, attached as Exhibit A. However, the DOHMH did not disclose its submission of the study to a peer-reviewed journal. After reading Dr. Bassett's Declaration, we made a second FOIL request, and DOHMH finally produced records relating to peer review under cover of a letter dated March 28, 2008. See Milburn letter of 2/20/08 and DOHMH letters of 3/4/08, 3/17/08 and 3/28/08, also attached as Exhibit A.

> problematic because of the lurking probability that people who look at caloric information are much different from everybody else, a priori. The fact that the chain under study was Subway is also a problem for interpretation because that chain is associated with lower-calorie meals and is marketed that way.
>
> In my mind, it is not possible to conclude that looking at the caloric info is causally related to calorie purchase . . . yes, there is an association, no doubt, but the association might just be reflecting the psychology of people who both look at caloric information and eat fewer calories. To establish causality, there would have to be other probably longitudinal study methods, like looking at consumption before and after caloric info were posted."

See Exhibit B, e-mail of 12/29/97. These comments of Dr. Shaw parallel points made by Dr. Allison in his two Declarations submitted in support of plaintiff's motion. Allison Declaration 1/29/08 at 22-28; Supplemental Allison Declaration 2/14/08 at 4-5.

5. In January 2008, the DOHMH submitted the report for publication in the *Journal of the American Medical Association* ("*JAMA*") as a "Brief Report." *JAMA* rejected the article without sending it out for peer review. See Exhibit C.

6. After the rejection by *JAMA*, Deputy Commissioner Bassett wrote to Dr. Mary Northridge. Drs. Bassett and Northridge are on the faculty together at the Columbia School of Public Health. Both are also on the Editorial Board of the *American Journal of Public Health* ("*AJPH*"). Dr. Northridge is the Editor-in-Chief of the *AJPH*, and Deputy Commissioner Bassett is an Associate Editor. Deputy Commissioner Bassett attached a summary of the DOHMH study and explained to Dr. Northridge that the study's content related to the DOHMH's effort to enact a regulation requiring posting of calorie information. Alluding to the decision enjoining the earlier version of Regulation 81.50, Deputy Commissioner Bassett wrote that "we [DOHMH] really want it [the study] published or at least accepted, in time for what we expect is coming—another suit." Dr. Northridge replied that she would "shepherd it through the peer review process once it is formally submitted" and requested Deputy Commissioner Bassett to list six

"suitable" peer reviewers. See Exhibit D.

7. Deputy Commissioner Bassett then wrote to Dr. Kelly Brownell, a Yale University obesity researcher who appeared in this action as *amicus curiae* in support of defendants in this action and NYSRA I. Deputy Commissioner Bassett asked Dr. Brownell if he would be willing to serve as a peer reviewer to review the DOHMH study for *AJPH*: "And as we are anxious for legal reasons (we expect another suit) to get it into print, would you be willing to turn a review around quickly?" Dr. Brownell agreed. Deputy Commissioner Bassett wrote to thank him and to advise him that he would be contacted by the *AJPH* to ask him to serve as a reviewer. See Exhibit E.

8. Deputy Commissioner Bassett made a similar request to another obesity researcher, Dr. Marion Nestle. Like Dr. Brownell, Dr. Nestle appeared in this action and in NYSRA I as an *amicus curiae*. Deputy Commissioner Barrett again alluded to the expected litigation and the consequent need to "turn [the review] around quickly." Again Deputy Commissioner Bassett first verified that Dr. Nestle "would be OK with it [serving as reviewer]" and then told Dr. Nestle that she would be contacted by *AJPH*. Deputy Commissioner Bassett also asked both Drs. Brownell and Nestle to suggest other reviewers. Dr. Nestle suggested "someone at CPSI?" CPSI, the Center for Science in the Public Interest, is yet another *amicus curiae* appearing on defendants' behalf that describes itself as an advocacy organization that has "led the advocacy efforts on behalf of New York City's restaurant calorie labeling rule." See Exhibit F.

9. I declare under penalty of perjury that the foregoing is true and correct.

Executed on April 2, 2008

Kent A. Yalowitz

ARNOLD & PORTER LLP

Nancy G. Millburn
Nancy_Milburn@aporter.com
212.715.1008
212.715.1399 Fax
399 Park Avenue
New York, NY 10022-4690

November 15, 2007

The City of New York
Department of Health and Mental Hygiene
General Counsel's Office
Attn: Rena Bryant
125 Worth Street
Room 602
New York, NY 10013

Re: Freedom of Information Law Request

Dear Records Access Officer:

This is a request pursuant to the New York State Freedom of Information Law, Public Officers Law Article 6, §§ 84-90 ("FOIL").

On October 24, 2007, the New York City Board of Health released a "Notice of Intention to Repeal and Reenact §81.50 of the New York City Health Code" (the "Notice"), available at http://www.nyc.gov/html/doh/downloads/pdf/public/notice-intention-hc-art81-50-1007.pdf. In the Notice, the Board of Health describes the following survey conducted by the New York City Department of Health and Mental Hygiene (the "Department"):

> To obtain more information about patterns of food consumption in New York City restaurant chains affected by the December 2006 Health Code Amendment, the Department conducted a large survey in a representative sample of major restaurants chains in New York City. The survey, conducted in March through June of this year, collected information from 11,835 diners at a random sample of 275 restaurants, representing 13 restaurant chains. As patrons left the restaurant, they were asked to supply their restaurant receipt and to answer several brief questions, including details of the purchase not reported on the receipt, whether the purchase was only for themselves,

whether they saw or used any available calorie information, and if so, whether this affected their purchase.

Notice, at 5.

I request all "records" within the custody, possession, or control of the Department, as the term "records" is defined in Public Officers Law § 86, that relate, in whole or in part, to the survey referenced above in the Notice. By way of example only, my request includes, but is not limited to, all records relating to the survey's design and development, any instruction(s) given to canvassers, all raw data collected during the survey (i.e., all questionnaires with information collected from the surveyed diners), and all qualitative or quantitative analyses performed on the raw data.

In accordance with the 2005 amendments to FOIL (Chapter 22, Laws of 2005), I look forward to receiving these records within twenty business days. You are authorized in advance to incur up to $1,000 in copying costs. If the amount will exceed that, please inform me. Please also call me if you have any questions about this request.

Sincerely,

Nancy G. Milburn

Nancy G. Milburn

THE CITY OF NEW YORK
DEPARTMENT OF HEALTH AND MENTAL HYGIENE
Michael R. Bloomberg
Mayor
Thomas R. Frieden, M.D., M.P.H.
Commissioner
nyc.gov/health

December 6, 2007

Arnold & Porter LLP
399 Park Avenue
New York, N.Y. 10022-4690
ATTENTION: Nancy G. Milburn, Esq.

Re: DOHMH Survey March-June, 2007
Control #: 2007FR02952

Dear Ms. Milburn:

Your Freedom of Information Law request for all "records" maintained by the Department of Health and Mental Hygiene regarding the above referenced survey is hereby granted in part and denied in part.

It is granted to the extent that you may have copies of the questionnaires with information collected from the surveyed diners, consisting of 23,680 pages (a questionnaire and the diner's receipt; and 275 observation logs (one per site). Also available are 184 pages of printed tables. These 24,137 paper records are available for a copying fee of $0.25/page. Also available are various data bases copied electronically onto a CD at $15.00 for the disc. If you wish these records, please forward a check payable to the New York City Department of Health and Mental Hygiene in the amount of $6,049.25.

Your request for copies of records relating to the survey's design and development and instructions given to canvassers is denied, pursuant to Section 87(2)(g) of the New York Public Officers Law since these records are inter-agency materials that are not instructions to staff that affect the public and/or final agency policy or determinations.

You may file a written appeal of the part of this decision denying your request within 30 days of the date of this letter. The appeal should be addressed to the Appeals Officer, Thomas Merrill, General Counsel, Department of Health and Mental Hygiene, 125 Worth Street (CN-30), New York, NY 10013. The Notice of Appeal should include the above control number, the date of this denial letter, a description of the records which were the subject for the request, and the full name and address of the requester.

Sincerely,

Rena Bryant

Rena Bryant
Records Access Officer

ARNOLD & PORTER LLP

Nancy G. Milburn
Nancy_Milburn@aporter.com
212.715.1008
212.715.1399 Fax
399 Park Avenue
New York, NY 10022-4690

December 28, 2007

The City of New York
Department of Health and Mental Hygiene
General Counsel's Office
Attn: Thomas Merrill
125 Worth Street (CN-30)
New York, NY 10013

Re: Appeal from Denial of Freedom of Information Law Request, Control No. 2007FR02952

Dear Mr. Merrill:

I write in response to Rena Bryant's letter, dated December 6, 2007, denying my request for records relating to the design and development of the diner survey and instructions given to canvassers.

The Board's decision to withhold these records from the public is without basis. The Board published the results of its diner survey in the Notice of Intention to Repeal and Reenact § 81.50 of the New York City Health Code, and it relies on the results of the survey as a basis for proposing the new Regulation. Having published the results of the survey and a description of how it was conducted, the Board should not be entitled to keep the survey design from the public. In the Notice of Intention, the Board is asking the public to accept the survey's findings, and the Board's interpretation of those findings. Under those circumstances, surely the public should be allowed to see how the City constructed the survey.

Moreover, the applicable case law and advisory opinions issued by the New York Committee on Open Government fully support disclosure of the withheld design materials. Records that represent a final agency determination or policy may not be withheld as "inter-agency or intra-agency material." N.Y. Pub. Officers Law Art. 6 §87(2)(g)(iii). Therefore, all records relating to the final proposed survey design—which was eventually adopted and implemented—should be disclosed pursuant to §87(2)(g)(iii). Even non-final agency records must be disclosed insofar as those records include statistical or factual data, pursuant to §87(2)(g)(i). "Factual data" means "objective information, in contrast to opinions, ideas or advice exchanged as part of the consultative or deliberative process of government decision making." Matter of Gould v. New York City Police Dept., 89 N.Y.2d 267, 276-77 (1996). The exemption in §87(2)(g)(i) permits

Records relating to canvassing instructions must be disclosed pursuant to §87(2)(g)(ii) because they are "instructions to staff that affect the public." In Advisory Opinion No. 12243 issued by the New York Committee on Open Government on July 31, 2000, Executive Director Robert Freedman clarified the "instruction to staff" exception:

> *§87(2)(g) is intended to [e]nsure that any so-called 'secret law' of an agency be made available, such as the policy 'upon which an agency relies' in carrying out its duties. Typically, agency guidelines, procedures, staff manuals and the like provide direction to an agency's employees regarding the means by which they perform their duties. Some may be 'internal,' in that they deal solely with the relationship between an agency and its staff. Others may provide direction in terms of the manner in which staff performs its duties in relation to or that affects the public, which would ordinarily be public. To be distinguished would be advice, opinions or recommendations that may be accepted or rejected. An instruction to staff, a policy or a determination each would represent a matter that is mandatory or which represents a final step in the decision making process.* (Emphasis added.)

As Mr. Freedman's opinion makes clear, directions to an agency's employees regarding the means by which they perform their duties must be disclosed under the exceptions in §87(2)(g), and an instruction to staff represents a mandatory direction. Our request for instructions to canvassers falls squarely within §87(2)(g)(ii) since such instructions directly affect how the canvassers related to and interacted with the public (e.g., what questions canvassers are to ask, which customers are to be polled, and which restaurants are selected for review).

FOIL is based upon a presumption of access. *See* N.Y. Pub. Officers Law Art. 6 §84. Courts have routinely held that the "exemptions [to disclosure] are to be narrowly construed, with the burden resting on the agency to demonstrate that the requested material indeed qualifies for exemption." Matter of Hanig v. State of N.Y. Dept. of Motor Vehs., 79 N.Y.2d 106, 109 (1992). The Department's expansive reading of §87(2)(g) is inconsistent with the principles underlying the New York State Freedom of Information Law. Therefore we request that all withheld records concerning the design and development of the survey, and instructions to canvassers be made available.

Sincerely,

Nancy G. Milburn

Nancy G. Milburn

cc: Mark W. Muschenheim, Esq.

THE CITY OF NEW YORK
DEPARTMENT OF HEALTH AND MENTAL HYGIENE
Michael R. Bloomberg
Mayor
Thomas R. Frieden, M.D., M.P.H.
Commissioner
nyc.gov/health

January 8, 2008

Arnold & Porter LLP
399 Park Avenue
New York, N.Y. 10022-4690
ATTENTION: Nancy G. Milburn, Esq.

Re: DOHMH Survey March-June, 2007
Control #: 2007FR02952

Dear Ms. Milburn:

I am in receipt of your letter dated December 28, 2007, appealing the partial denial of your Freedom of Information Law request for all "records" maintained by the Department of Health and Mental Hygiene regarding the above referenced survey.

Please be advised that your appeal is herewith granted. Records related to the survey's design and development and the instructions given to canvassers will be added to the materials put on a CD, as described in Ms. Bryant's December 6, 2007 letter. We are also in receipt of your December 21, 2007 request for certain paper documents, and indicating that you do not want copies of the individual questionnaires and receipts. Please forward a check to the attention of Rena Bryant at the address below, payable to the New York City Department of Health and Mental Hygiene in the amount of $129.75, covering the costs of copying both 184 pages of printed tables and 275 pages of observation logs (459 pages at $0.25/page=$114.75) and the CD containing the data bases and survey materials (at $15.00 for the disc).

Yours truly,

Thomas Merrill
General Counsel
Appeals Officer

ARNOLD & PORTER LLP

Nancy G. Milburn
Nancy_Milburn@aporter.com
212.715.1008
212.715.1399 Fax
399 Park Avenue
New York, NY 10022-4690

February 20, 2008

The City of New York
Department of Health and Mental Hygiene
General Counsel's Office
Attn: Thomas Merrill
Rena Bryant
125 Worth Street - Room 602
New York, NY 10013

Re: **FOIL Request, Control No. 2007FR02952**

Dear Mr. Merrill and Ms. Bryant:

On November 15, 2007 I made a request under the Freedom Of Information Law ("FOIL") for records relating to a survey of restaurants conducted by the NYC Department of Health and Mental Hygiene (the "Department") in 2007 (the "survey"). A copy of that request is enclosed. On December 6, 2007 you granted the request in part. Following an appeal on January 8, 2008, you granted the request in full, and provided us with responsive documents. Copies of the partial and full grant letters are enclosed.

On February 8, 2008, Commissioner Thomas Frieden and Deputy Commissioner Mary Bassett signed Declarations in the case, *New York State Restaurant Association v. New York City Board of Health*, 08-Civ-1000 (RJH), that refer to information and records related to the survey that have not been provided to me. Copies of those Declarations are enclosed. This is to request that the Department provide to me the following documents:

(a) All records indicating that, at the time of the study, Subway restaurants posted nutritional information "on a sticker placed on a display case near the cash register." *See* Frieden Declaration ¶ 32;

(b) All records indicating that, at the time of the survey, Subway restaurants posted calorie information on glass case decals. *See* Bassett Declaration ¶ 8.

(c) All records consisting of, relating to or referring to, manuscripts or articles summarizing the methods and results of the survey prepared for publication, including without limitation any correspondence with journals or journal editors regarding possible publication. *See* Bassett Declaration ¶¶ 4-5.

(d) All other records specified in my November 15, 2007 FOIL request that have not yet been produced to us (aside from the questionnaires and receipts we advised you we did not want).

In accordance with the 2005 amendments to FOIL (Chapter 22, Laws of 2005), the requested documents should have already been provided to me. Please provide them to me as soon as possible. You are authorized in advance to incur up to $500 in copying costs. If the amount will exceed that, please inform me. Please also call me if you have any questions about this request.

Sincerely,

Nancy G. Milburn

Nancy G. Milburn

Enclosures

cc: Mark Muschenheim, Esq.

THE CITY OF NEW YORK
DEPARTMENT OF HEALTH AND MENTAL HYGIENE

Michael R. Bloomberg
Mayor

Thomas R. Frieden, M.D., M.P.H.
Commissioner

nyc.gov/health

March 4, 2008

Arnold & Porter LLP
399 Park Avenue
New York, N.Y. 10022-4690
ATTENTION: Nancy G. Milburn, Esq.

Re: Control #: 2008FR00493

Dear Ms. Milburn:

I am in receipt of your letter dated February 20, 2008, requesting additional records pursuant to the Freedom of Information Law maintained by the Department of Health and Mental Hygiene (the "Department"), regarding findings at Subway restaurants and "all records consisting of … methods and results of the survey prepared for publication, including without limitation any correspondence with journals or journal editors regarding possible publication." In your letter you cite to the declarations of Dr. Frieden and Dr. Bassett in connection with the litigation you commenced in federal court. You also request "all other records specified in [your] … November 15, 2007 FOIL request that have not yet been produced to us (aside from the questionnaires and receipts we advised you we did not want)" under FOIL Control #2007FR02952.

Please be advised that the Department considers your initial FOIL request, Control #2007FR02952, as having been fully complied with. With respect to the specific records you request in your February 20, 2008 letter, please be further advised that the Department has undertaken a search to determine if there are any records responsive to your request, and we will notify you of any available records after such a search is completed.

Yours truly,

Rena Bryant

Rena Bryant
Records Access Officer

THE CITY OF NEW YORK
DEPARTMENT OF HEALTH AND MENTAL HYGIENE
Michael R. Bloomberg
Mayor
Thomas R. Frieden, M.D., M.P.H.
Commissioner
nyc.gov/health

March 17, 2008

Arnold & Porter LLP
399 Park Avenue
New York, N.Y. 10022-4690
ATTENTION: Nancy G. Milburn, Esq.

Re: Control No.: 2008FR00493

Dear Ms. Milburn:

Please be advised, in response to your letter dated February 20, 2008, pursuant to the Freedom of Information Law (NY Public Officers Law §§84-90), requesting additional records maintained by the New York City Department of Health and Mental Hygiene (Department), regarding findings at Subway restaurants and "all records consisting of … methods and results of the survey prepared for publication, including without limitation any correspondence with journals or journal editors regarding possible publication" and "all other records specified in [your] … November 15, 2007 FOIL request that have not yet been produced to us (aside from the questionnaires and receipts we advised you we did not want)" under FOIL Control No. 2007FR02952, that there are no additional records.

With respect to records related to the declarations of Dr. Frieden and Dr. Bassett in connection with the litigation you commenced in federal court, the Department has identified 56 pages of records responsive to your request. These records have been redacted of intra-agency information that are not final agency determinations. See, NY Public Officers Law § 87(2)(g). If you wish copies of these records, please forward a check payable to the New York City Department of Health and Mental Hygiene in the amount of $ 14.00 to my attention.

Yours truly,

Rena Bryant

Rena Bryant
Secretary and Records Access Officer

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THE CITY OF NEW YORK
DEPARTMENT OF HEALTH AND MENTAL HYGIENE
Michael R. Bloomberg
Mayor
Thomas R. Frieden, M.D., M.P.H.
Commissioner
nyc.gov/health

March 28, 2008

Arnold & Porter LLP
399 Park Avenue
New York, N.Y. 10022-4690
ATTENTION: Nancy G. Milburn, Esq.

Re: Control No.: 2008FR00493

Dear Ms. Milburn:

As per the Department's March 17, 2008 letter to you, attached please find 46 pages of responsive and releasable records regarding the above-referenced FOIL request. Please note that, due to a typographical error in the Department's March 17, 2008 letter to you, the actual number of pages being released is 46, not 56. Please advise whether you wish to seek a refund from the Department for this difference ($ 2.50).

Sincerely,

Rena Bryant

Rena Bryant
Records Access Officer

Attachments

2008FR00493A&P3.doc

From: Anna Caffarelli
Sent: Thursday, March 13, 2008 3:33 PM
To:
Subject: FW: Potential MMWR on calorie information

From: Thomas R. Frieden
Sent: Thursday, March 13, 2008 3:32 PM
To: Anna Caffarelli
Subject: FW: Potential MMWR on calorie information

From: Thomas R. Frieden
Sent: Friday, January 04, 2008 11:40 AM
To: 'Shaw, Frederic (CDC/CCHIS/NCHM)'; Weatherwax, Douglas (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR on calorie information

Fred,

Thanks for your comments. Although we recognize the limitations of the analysis, we don't agree on your critique – but we can agree to disagree.

At this point we'll move on for this project, but keep MMWR in mind for future initiatives.

All the best,

Tom

From: Shaw, Frederic (CDC/CCHIS/NCHM) [mailto:fxs6@CDC.GOV]
Sent: Saturday, December 29, 2007 1:38 PM
To: Thomas R. Frieden; Weatherwax, Douglas (CDC/CCHIS/NCHM)
Cc: Shaw, Frederic (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR on calorie information

Tom,

Sorry it took me a bit to get back to you. I read the article carefully and asked a few others to also take a look, and I do not feel it bears publishing in MMWR as it stands. The conclusions being drawn by the study are, of course, problematic because of the lurking probability that people who look at caloric information are much different from everybody else, a priori. The fact that the chain under study was Subway is also a problem for interpretation because that chain is associated with lower-calorie meals and is marketed that way.

In my mind, it is not possible to conclude that looking at the caloric info is causally related to reduced calorie purchase. yes, there is an association, no doubt, but the association might just be reflecting the psychology of people who both look at caloric information and eat fewer calories. To establish causality, there would have to be other probably longitudinal study methods, like looking at consumption before and after caloric info were posted.

I am not saying that this cannot be fixed scientifically somehow in the report, but the problem is that MMWR is not a good format for presenting epi data for which the interpretation might be controversial or complex, especially

related to confounding. There just is not enough space in 1400 words to discuss the possible problems with interpretation.

One other thought: we were all intrigued by the caloric intake info itself, like the distribution of caloric intakes per meal etc. One reviewer said this alone might make a good MMWR and I tend to agree. You could just present the distribution of caloric intake and try to analyze what foods were the main high calorie culprits. This would be interesting and would probably make a nice MMWR because it would be fairly simple and would not try to make causal associations with looking at caloric info posted, and it could carry a pretty clean public health message--like x% of the total calorie intake at these places can be avoided by doing y.

One other small note. I am very impressed with what you are trying to do in NYC with obesity. I think many of us are, and I applaud you for thinking out of the box and taking some unpopular stands. We are interested in publishing more of your studies in MMWR, with the caveats above.

Happy New year to you,

Fred

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Tuesday, December 18, 2007 4:46 PM
To: Weatherwax, Douglas (CDC/CCHIS/NCHM)
Cc: Shaw, Frederic (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR on calorie information

Doug,

Hope you are well. Any way to get a sense of how this is going or when we might hear something?

Thanks.

Tom

From: Thomas R. Frieden
Sent: Tuesday, November 27, 2007 2:13 PM
To: Thomas R. Frieden; 'Weatherwax, Douglas (CDC/CCHIS/NCHM)'
Cc: 'Shaw, Frederic (CDC/CCHIS/NCHM)'
Subject: RE: Potential MMWR on calorie information

Doug,

Resending along with the table.

Thanks.

Tom

From: Thomas R. Frieden
Sent: Tuesday, November 27, 2007 2:12 PM
To: 'Weatherwax, Douglas (CDC/CCHIS/NCHM)'
Cc: 'Shaw, Frederic (CDC/CCHIS/NCHM)'
Subject: RE: Potential MMWR on calorie information

Doug,

Efforts to address the serious and progressive epidemic of obesity are hampered by lack of information on the impact of various interventions. Some restaurants provide information on calories, but whether customers see that information and whether seeing information affects purchases has not been well studied. We recently conducted a large cross-sectional study of customers who had just purchased food from fast-food restaurants.

In brief, the study showed that less than 5% of patrons reported seeing calorie information except at one fast-food chain (Chain A), which displayed calorie information somewhat prominently. At Chain A (Subway), 31% saw the information, and those who reported seeing calorie information purchased 49 fewer calories than those who did not; those reporting that calorie information influenced them purchased 92 fewer calories than those who saw the information but said it did not influence them.

Of course, any cross-sectional survey has limitations. Chain A patrons could be more calorie-conscious, more likely to notice calorie information, or more likely to change purchase decisions based on calorie information. We have addressed these limitations in the editorial note.

In spite of these limitations, the information in this survey suggests that providing calorie information prominently may increase the proportion of restaurant customers who see the information and the proportion who reduce calorie purchases as a result. We know of no other survey of its kind, our sample was large and based on a random sample of restaurants, and non-participation in the survey appears to have been largely random. To the extent that our findings reflect restaurant patron behavior generally, prominent posting of calorie information could substantially reduce calorie consumption, overweight and obesity.

All the best,

Tom

From: Weatherwax, Douglas (CDC/CCHIS/NCHM) [mailto:aoe8@CDC.GOV]
Sent: Wednesday, November 14, 2007 8:35 AM
To: Thomas R. Frieden
Cc: Saundra Welch; Judith Freeman
Subject: RE: Potential MMWR

Attached is a copy of MMWR Made Simple, which I pass out to new EISOs each year and which you might find helpful.
Doug

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Tuesday, November 13, 2007 2:10 PM
To: Weatherwax, Douglas (CDC/CCHIS/NCHM)
Cc: Saundra Welch; Judith Freeman
Subject: RE: Potential MMWR

Thanks, I'll call you then.

From: Weatherwax, Douglas (CDC/CCHIS/NCHM) [mailto:aoe8@CDC.GOV]
Sent: Tuesday, November 13, 2007 1:38 PM
To: Thomas R. Frieden
Cc: Saundra Welch; Judith Freeman
Subject: RE: Potential MMWR

404-498-2365. 8:30 will be fine.

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Tuesday, November 13, 2007 11:58 AM
To: Weatherwax, Douglas (CDC/CCHIS/NCHM)
Cc: Saundra Welch; Judith Freeman
Subject: RE: Potential MMWR

8:30 tomorrow? What number would be good to call you at?

From: Weatherwax, Douglas (CDC/CCHIS/NCHM) [mailto:aoe8@CDC.GOV]
Sent: Tuesday, November 13, 2007 11:50 AM
To: Thomas R. Frieden
Cc: Mary T Bassett
Subject: RE: Potential MMWR

Tomorrow morning, I'll be available from 8 to 10. Also, Thursday morning from 8 to 9.

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Tuesday, November 13, 2007 11:33 AM
To: Weatherwax, Douglas (CDC/CCHIS/NCHM)
Cc: Mary T Bassett
Subject: Re: Potential MMWR

Understand completely. Is there a time we could chat briefly in the next few days?

----- Original Message -----
From: Weatherwax, Douglas (CDC/CCHIS/NCHM) <aoe8@CDC.GOV>
To: Thomas R. Frieden
Cc: Mary T Bassett
Sent: Tue Nov 13 08:19:04 2007
Subject: RE: Potential MMWR

Tom,
How fast we can schedule and publish will depend upon how fast the report is cleared and whether it is accepted for publication by the Editor.
Doug

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Tuesday, November 13, 2007 8:10 AM
To: Weatherwax, Douglas (CDC/CCHIS/NCHM)
Cc: Mary T Bassett
Subject: RE: Potential MMWR

Doug,

Many thanks for your guidance on this. Perhaps we can chat later this week.

A simple question, though…is there any way we might get this out by mid-January?

Thanks.

Tom

From: Shaw, Frederic (CDC/CCHIS/NCHM) [mailto:fxs6@CDC.GOV]
Sent: Monday, November 12, 2007 3:19 PM
To: Thomas R. Frieden
Cc: Weatherwax, Douglas (CDC/CCHIS/NCHM); Hewitt, Suzanne M. (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR

Tom,

MMWR would be interested in receiving this report.

Please ask the authors to contact Doug Weatherwax at MMWR (who is cc'd on this email) to get further advice on submission of the manuscript and scheduling. Please also follow the guidance for MMWR contributors on the MMWR web site for contributors: http://www.cdc.gov/mmwr/author_guide.html <http://www.cdc.gov/mmwr/author_guide.html> .

Thanks for considering MMWR for your publication.

Also, please feel free to write or call me directly anytime.

Warm regards,

Fred

Frederic E. Shaw, MD, JD

Editor, MMWR Series
MMWR Office, NCHM, CoCHIS
Centers for Disease Control and Prevention
1600 Clifton Rd. NE, MS E-90
Atlanta, GA 30333
Voice: (404) 498-6364
Fax: (404) 498-2389

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Tuesday, November 06, 2007 2:51 PM
To: Shaw, Frederic (CDC/CCHIS/NCHM)
Cc: Weatherwax, Douglas (CDC/CCHIS/NCHM); Hewitt, Suzanne M. (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR

Fred,

We would just report the association. One could argue that those who were health-conscious were more likely to see the information. But at the one chain that published information prominently (which is the largest meal chain in NYC), 31% of people saw it, so it's hardly a small group. We asked people whether seeing calorie information changed their choices. Those who saw but said it had not affected them bought about the same amount (13 calories less). That suggests that all types of people will see it, but the information will influence the actions of some more than others. If calorie information is prominent enough you don't have to be different to see it.

The chain at that time had provided the information in a prominent manner but not nearly as prominent as what we have mandated. This suggests to us that providing the information in all chains in an even more prominent manner would certainly get more people to see it, and that some portion of them would be likely to reduce their caloric consumption.

Thanks.

Tom

From: Shaw, Frederic (CDC/CCHIS/NCHM) [mailto:fxs6@CDC.GOV]
Sent: Tuesday, November 06, 2007 1:06 PM
To: Thomas R. Frieden
Cc: Weatherwax, Douglas (CDC/CCHIS/NCHM); Hewitt, Suzanne M. (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR

Tom, do you intend that the report will rest on the conclusion that seeing the calorie information caused fewer calories to be purchased? That would be very hard to substantiate, would it not, because there are lots of reasons why persons who saw

caloric information might purchase fewer calories. People who look at calorie information no doubt are fundamentally different than people who do not. Can you adjust for these factors somehow?

Fred

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Monday, November 05, 2007 5:53 PM
To: Shaw, Frederic (CDC/CCHIS/NCHM)
Cc: Weatherwax, Douglas (CDC/CCHIS/NCHM); Hewitt, Suzanne M. (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR

Fred,

Here are the basics, attached and pasted below. It doesn't get into either limitations or implications, but does give the basics of the study and findings. Tomorrow's officially a City holiday, so would be a quiet time for a chat if it works for you. We spoke with Jennifer Seymour and two of her colleagues at Nutrition (Acting ADS and science officer).

All the best,

Tom

Background: Fast-food consumption has been linked to increased calorie intake and obesity. We conducted a cross-sectional survey of fast-food patrons to establish calories consumed per purchase, proportion seeing calorie information, and effect of seeing calorie information on calories purchased. One of the 11 fast-food chains sampled displayed calorie information conspicuously at point-of-purchase.

Methods: From March through June 2007 we administered surveys to adult patrons exiting a random sample of fast-food chains throughout New York City. Survey questions assessed food and beverage purchases, condiments added, observation of calorie information and, if it was seen, whether patrons reported that they used calorie information. We collected receipts to calculate total calories using published values.

Results: Overall, 11,829 receipts were collected from 273 sites. Customers purchased a mean of 824 calories (range, 303–1350); >30% purchased $\geq$1,000 calories. Only 918 (8%) patrons reported seeing calorie information. Patrons of the chain posting calorie information were significantly more likely to see nutritional information than other patrons (32% vs. 4%, $p<0.001$). Among this chain's patrons, those seeing calorie information purchased 47 fewer calories than those not seeing the information (688 calories vs. 735 calories, $p=0.006$). Patrons who reported both seeing and using calorie information purchased 108 fewer calories than those not seeing the information (627 calories vs. 735 calories, $p<0.001$).

Conclusions: Calorie information was not seen by >95% of restaurant customers at 10 chains, even after food purchase. Patrons at the one chain which displayed information prominently at the point-of-purchase were much more likely to see calorie information, and those who saw calorie information purchased fewer calories.

From: Shaw, Frederic (CDC/CCHIS/NCHM) [mailto:fxs6@CDC.GOV]
Sent: Monday, November 05, 2007 6:24 AM
To: Thomas R. Frieden
Cc: Weatherwax, Douglas (CDC/CCHIS/NCHM); Hewitt, Suzanne M. (CDC/CCHIS/NCHM)
Subject: RE: Potential MMWR

Tom,

Yes we would be happy to discuss. Sounds interesting.

Could you ask your folks to put together a little 250-abstract with the methods, and the major findings laid out (something a little more expanded from the paragraph below) and get it back to me before we talk. Also, who did you speak with at Nutrition?

I am working off site this week, but available at 404 538 3967.

Thanks for thinking of MMWR.

Best regards,

Fred

Frederic E. Shaw, MD, JD
Editor, MMWR Series
MMWR Office, NCHM, CoCHIS
Centers for Disease Control and Prevention
1600 Clifton Rd. NE, MS E-90
Atlanta, GA 30333
Voice: (404) 498-6364
Fax: (404) 498-2389

From: Thomas R. Frieden [mailto:tfrieden@health.nyc.gov]
Sent: Saturday, November 03, 2007 2:05 PM
To: Shaw, Frederic (CDC/CCHIS/NCHM)
Subject: Potential MMWR

[redacted] [redacted] [redacted] [redacted]

From: Cheryl de Jong-Lambert <cdejongl@health.nyc.gov> **Sent:** Thu 01/10/2008 04:47 PM
To: Sekai Chideya <schideya@health.nyc.gov>
CC: "Thomas R. Frieden" <tfrieden@health.nyc.gov>,Mary T Bassett <mbassett@health.nyc.gov>,Lynn Silver <lsilver@health.nyc.gov>,Tamara Dumanovsky <tdumanov@health.nyc.gov>,Christina Huang <chuang@health.nyc.gov>,Cathy Nonas <cnonas@health.nyc.gov>,Thomas Matte <tmatte@health.nyc.gov>,youngcr1@verizon.net
Subject: RE: Report for submission to JAMA
Attachments:

Congratulations! I'm currently on the JAMA site for a resubmit. The table may need to be uploaded separately, but other than that it looks like you've got everything. I can submit it tomorrow, following up with any questions, and then cross my fingers for both of these.

Cheryl

From: Sekai Chideya
Sent: Thursday, January 10, 2008 4:35 PM
To: Cheryl de Jong-Lambert
Cc: Thomas R. Frieden; Mary T Bassett; Lynn Silver; Tamara Dumanovsky; Christina Huang; Cathy Nonas; Thomas Matte; youngcr1@verizon.net
Subject: Report for submission to JAMA

Hi Cheryl,

The calorie labeling report that Mary Bassett's division has been writing is ready for submission to JAMA as a "Brief Report." TRF mentioned that you could assist us with this process, since he's being listed as the corresponding author.

I'm attaching the manuscript and cover letter, but I am not sure how you'd like to proceed, or what else you might need? Please let me know.

Thanks so much,

Sekai

Sekai Chideya, MD, MPH
Director of Special Projects
New York City Department of Health and Mental Hygiene
125 Worth Street, Room 349
New York, NY 10013
Office: (212) 788-5330
Cell: (917) 709-2142
Fax: (212) 964-40472
email: schideya@health.nyc.gov

Archive Mail Information

Archive Mail Information

[redacted]

From: Mary E Northridge <men11@columbia.edu>
Sent: Tue 01/15/2008 06:00 PM
To: Mary T Bassett <mbassett@health.nyc.gov>
CC:
Subject: Re: trying to get something published in in the nxt 6 months
Attachments:

Mary B.: Our time to publication is way down, given on-line publishing ahead of print. I will shepard it through the peer review process once it is formally submitted at submit.ajph.org. Kindly list 6 suitable peer referees at submission. How does this sound? Mary N.

On Tue, 15 Jan 2008, Mary T Bassett wrote:

> Mary,
>
>
>
> I am attaching a short piece on a survey of fast food chains in NYC. We
> submitted it to JAMA- but they rejected it without review.
>
>
> The content has relevance to our efforts to require posting of calorie
> information on menu boards here in NYC. The Board of Health passed a
> health code amendment about a year ago, but the NYS Restaurant
> Association successfully sued to have it overturned last summer. This
> survey shows 1) calories purchases during the lunch hour are pretty
> high, about 1/3 of purchases about 1000 cals, if you eat chicken it gets
> even higher; 2) nearly everyone who walks in the fast food chains walks
> out with no idea of the calorie content of their purchases; 3) At one
> chain - Subway- some calories info was posted before the DOHMH
> requirement, so we could look at its impact. Here 30% saw cal info and
> among those people who saw, especially those who see and use, purchase
> fewer calories.
>
>
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> We really want it published or at least accepted, in time for what we
> expect is coming- another suit. I think AJPH is a good place- but the
> time to print is pretty long., Any thoughts on whether this might fit
> with an upcoming theme issue- I know this is the fastest way into print.
>
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> MTB
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> **********
> The New York City Department of Health & Mental Hygiene is now offering information important for the health of all New Yorkers. To sign up for these new and valuable updates, log-on to our website at http://www.nyc.gov/health/email and select the NYC DOHMH updates you'd like to receive.
>
> IMPORTANT NOTICE: This email is meant only for the use of the intended recipient. It may contain confidential information that is legally privileged or otherwise protected by law. If you have received this communication in error, please notify me immediately by replying to this message and please delete it from your computer. Thank you for your cooperation.
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[redacted]

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>

>
>

[redacted]

From: Mary T Bassett <mbassett@health.nyc.gov> **Sent:** Wed 01/16/2008 11:50 PM
To: Kelly Brownell <kelly.brownell@yale.edu>
CC: Sekai Chideya <schideya@health.nyc.gov>
Subject: RE: would you be willing to serve as a reviewer?
Attachments:

Many thanks! You'll be contacted by the Journal to ask if you are willing to serve as a reviewer.

Sekai – please see suggestion of David Ludwig.

From: Kelly Brownell [mailto:kelly.brownell@yale.edu]
Sent: Wednesday, January 16, 2008 11:43 PM
To: Mary T Bassett
Subject: Re: would you be willing to serve as a reviewer?

yes. David Ludwig at Harvard would be a good reviewer as well. he has published on the topic

Kelly D. Brownell, Ph.D.
Professor of Psychology, Epidemiology and Public Health
Director, Rudd Center for Food Policy and Obesity
Yale University

Yale University - Rudd Center
309 Edwards Street - Box 208369
New Haven, CT 06520-8369
203-432-7790, 203-432-9674 (fax)
kelly.brownell@yale.edu, www.YaleRuddCenter.org

Mary T Bassett wrote:
Kelly, we are submitting an article based on a survey of patrons at NYC fast food chains to Am J Pub Health. Would you be willing to serve as a reviewer? And, as we are anxious for legal reasons (we expect another suit) to get it into print, would you be willing to turn a review around quickly? It is 1500 words.

Can you suggest another such reviewer?

I am copying Sekai Chideya who is coordinating the submission of the article

this communication in error, please notify me immediately by replying to this message and please delete it from your computer. Thank you for your cooperation.

[redacted]

From: Marion Nestle [mailto:marion.nestle@nyu.edu]
Sent: Wednesday, January 16, 2008 7:30 PM
To: Mary T Bassett
Subject: RE: may I suggest you as a reviewer?

Someone at CSPI?

From: Mary T Bassett [mailto:mbassett@health.nyc.gov]
Sent: Wednesday, January 16, 2008 5:30 PM
To: Marion Nestle
Subject: RE: may I suggest you as a reviewer?

Yes, I am suggesting you and you will be contacted by AJPH- but I wanted to be sure you would be OK with it. Can you suggest anyone else? I am sending a similar email to Kelly Brownell. Anyone else?

From: Marion Nestle [mailto:marion.nestle@nyu.edu]
Sent: Wednesday, January 16, 2008 7:26 PM
To: Mary T Bassett
Subject: RE: may I suggest you as a reviewer?

Sure. Is this being sent by the journal?

From: Mary T Bassett [mailto:mbassett@health.nyc.gov]
Sent: Wednesday, January 16, 2008 5:18 PM
To: marion.nestle@nyu.edu
Subject: may I suggest you as a reviewer?

We are submitting an article based on the receipt at fast food chains survey to Am J Pub Health. You did not participate in the analysis or write up- so I think it would be OK to ask you to serve as a reviewer. Would you be willing? And, as we are anxious for legal reasons (we expect another suit) to get it into print, would you be willing to turn it around quickly? It is 1500 words.

****

http://www.nyc.gov/health/email and select the NYC DOHMH updates you'd like to receive.

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Exhibit N

SENATE HEALTH COMMITTEE ANALYSIS
Senator Deborah V. Ortiz, Chair

BILL NO:	**SB 144**	**S B 1 4 4**
AUTHOR:	**Runner**	
AMENDED:	**As introduced**	
HEARING DATE:	**April 27, 2005**	
FISCAL:	**Appropriations**	

CONSULTANT:
Machi / ak

TESTIMONY TAKEN – VOTE ONLY

SUBJECT

Retail Food

SUMMARY

This bill repeals the California Uniform Retail Food Facilities Law (CURFFL) and establishes the California Retail Food Code (CalCode), entirely rewriting retail food facility statute.

ABSTRACT

Existing federal administrative action:
The United States Food and Drug Administration (FDA) drafts model food safety codes to assist state and local governments in initiating and maintaining effective programs for the prevention foodborne illness. The FDA first published the Model Food Code in 1993. Between 1993 and 2001 the Model Food Code has been updated and revised every two years.

Existing state law:
In 1985 the legislature consolidated the various overlapping laws that existed at that time into a comprehensive "California Uniform Retail Food Facilities Law" (CURFFL). CURFFL provides for the regulation of health and sanitation standards for retail food facilities by the State Department of Health Services (DHS) and is primarily enforced by local health agencies. CURFFL prohibits a food facility from opening for business without a valid permit issued by the local enforcement agency. A violation of any provision of CURFFL is a misdemeanor.

CURFFL
General Provisions

- Establishes authority of local environmental health jurisdictions to adopt a food safety inspection program with oversight by the DHS Food and Drug Branch.

Continued---

- Establishes uniform food safety and sanitation requirements for local jurisdictions to follow. Provisions are also provided to allow a local jurisdiction to adopt a grading system for food facilities, to prohibit any type of food facility, and to regulate the provision of toilet and hand washing facilities.
- Requires one certified food manager to pass an approved examination for each food facility handling unpackaged potentially hazardous foods with the exception of temporary food facilities and mobile food facilities.
- Grandfathers local jurisdictions that had a food handler training program in effect since 1998

Plan Review and Permits

- Requires plans for new and remodeled food facilities to be submitted and approved by the local environmental health jurisdiction prior to operation.
- Requires that a permit be issued for each food facility and has provisions for suspension, revocation and modification of that permit. Permit fees are established by each local jurisdiction.
- Provides provisions for penalties for operating without a permit.

Enforcement and Inspection

- Provides authority for the local jurisdiction to inspect, immediately suspend a permit, conduct hearings, take samples or other evidence, impound food or equipment, and issue reports.
- Provides provisions for misdemeanor penalties for violations.
- Makes the owner, manager, or operator of the food facility responsible for violations.
- Establishes criteria for food facility inspection format, standardization of inspections, and Internet reporting.

Temperature Control

- Establishes standards for temperature control of potentially hazardous foods. Establishes provisions for using time as a public health control for potentially hazardous foods.
- Contains exemptions from temperature holding requirements for Korean Rice Cakes and Chinese-style Roast Duck.
- Establishes cooking standards for certain potentially hazardous foods.
- Establishes standards for thawing, reheating and cooling of potentially hazardous foods.

Protection from Contamination/Adulteration

- Requires that food be protected from contamination, obtained from approved food sources and inspected upon receipt.
- Contains provisions for use of a Hazard Analysis Critical Control Point Plan under certain conditions.
- Requires that food facilities be constructed and operated to exclude vermin.
- Excludes live animals, birds or fowl. Exceptions are provided for service animals, dogs under the control of a uniformed law enforcement officer, and aquariums and enclosed aviaries that do not create a public health problem.

Continued---

- Establishes standards for sanitization of food contact surfaces.

- Prohibits sulfites on potentially hazardous foods.
- Establishes standards for food storage and display.
- Establishes standards for water potability and temperature.

Employee Health and Hygiene

- Establishes requirements for food handler health and hygiene.
- Requires that signs be posted for hand washing, no smoking, review of the last inspection report availability, and if no toilet facilities are available to the public if the facility provides table service.

Sanitation

- Restricts sleeping accommodations and sale or service of foods prepared in a private home.
- Contains sanitary provisions for equipment, utensils, linens, facilities, and proper disposal of waste material.

Facilities and Equipment

- Establishes standards for food service equipment.
- Contains provisions for installation and maintenance of plumbing, employee changing rooms, hand washing sinks, janitorial sinks, utensil washing sinks, and employee and customer toilet facilities.
- Contains provisions for ventilation of cooking equipment, comfort, and toilet rooms.
- Requires enclosure of food establishments with exceptions for produce stands, swap meet prepackaged food stands, dining areas, open-air barbecues and wood burning ovens, beverage bars, The Mercado La Paloma, and certain outdoor displays and beverage dispensing operations.
- Contains provisions for construction of floors, walls and ceilings of food facilities.
- Contains a separate article with separate requirements for open-air barbecue facilities, vending machines, mobile food facilities, mobile food preparation and stationary mobile food preparation units, commissaries, temporary food facilities, nonprofit charitable temporary food facilities, produce stands, certified farmers markets, swap meet prepackaged food stands, satellite food distribution facilities, restricted food service transient occupancy establishments and agricultural home stays, and food facility donations.

Exemptions for child day care facilities, community care facilities, and residential care facilities for the elderly

- A specific exemption from CURFFL is provided for child day care facilities, community care facilities, and residential care facilities for the elderly.

This bill:

Would repeal CURFFL and would recast and revise the provision of California state law, regulating the retail sale of food, by creating the California Retail Food Code (CalCode).

Continued---

Below is a list of some of the enhancements to California law made in the CalCode:

Documentation

- Introduces the concept of identifying a "person in charge" at a retail food facility and specifies the food safety duties and qualifications of said person.
- Requires operators of temporary food facilities and mobile food facilities to demonstrate knowledge of food safety practices.
- A public health reasons annex will be prepared for the CalCode to provide the public health significance of the sections of the CalCode. The annex will be modeled after the public health reasons annex of the model FDA model Food Code.

Uniformity and Consistency

- Consolidates food safety requirements found in CURFFL articles 9-20 in order to ensure uniformity in interpretation and application of law.
- Clarifies the enforcement actions that local health officials should take relative to temporary food facilities and activities.
- Provides clear, uniform requirements for outdoor food service.
- Requires food labeling that is in conformance with California's Sherman Food, Drug, and Cosmetic Law.
- Contains provisions that will clarify and expand the requirements pertaining to equipment, utensils, and linens used in retail food service operations.

Best Available Science

- Includes requirements applicable to the transportation of food.
- Expands the requirements related to the minimum cooking temperatures applicable to different types of foods.
- Expands the type of food preparation activities that can take place on a "mobile food facility," consistent with good public health principles.
- Expands and clarifies the restrictions and requirements related to utilization of Hazard Analysis Critical Control Plans.

At Risk Populations

- Contains specific provisions to address food service at "Supervised Care Facilities" (i.e., community care facilities, residential care facilities for the elderly, and child day care facilities) within the scope of its regulatory provisions.

Flexibility

- Contains detailed "variance" provisions, thereby providing for alternate food safety practices that provide equivalent consumer protection.

Significant provisions within the CalCode include the following:

General Provisions

- Establishes authority of local environmental health jurisdictions to adopt a food safety inspection program with oversight by the California Department of Health Services Food and Drug Branch.

Continued---

- Establishes uniform food safety and sanitation requirements for local jurisdictions to follow. Provisions are also provided to allow a local jurisdiction to adopt a grading system for food facilities, to prohibit any type of food facility, and to regulate the provision of toilet and hand washing facilities.
- Requires one certified food manager to pass an approved examination for each food facility handling unpackaged potentially hazardous foods.
- Grandfathers local jurisdictions that had a food handler training program in effect since 1998.

Plan Review and Permits

- Requires plans for new and remodeled food facilities to be submitted and approved by the local environmental health jurisdiction prior to operation.
- Requires that a permit be issued for each food facility and has provisions for suspension, revocation and modification of that permit. Permit fees are established by each local jurisdiction.
- Requires registration of supervised care facilities where food service is provided for less than 15 persons.
- Provides provision for penalties for operating without a permit.

Enforcement and Inspection

- Provides authority for the local jurisdiction to inspect, immediately suspend a permit, conduct hearings, take samples or other evidence, impound food or equipment, and issue reports.
- Provides provisions for misdemeanor penalties for violations.
- Makes the owner, manager, or operator of the food facility responsible for violations.
- Establishes criteria for food facility inspection format, standardization of inspections, and Internet reporting.

Temperature Control

- Establishes standards for thawing and hot and cold holding of potentially hazardous foods.
- Establishes provisions for using time as a public health control for potentially hazardous foods.
- Contains exemptions from temperature holding requirements for Korean Rice Cakes and Chinese-style Roast Duck.
- Establishes and expands cooking standards for certain potentially hazardous foods.
- Establishes standards for reheating and cooling potentially hazardous foods.

Protection from Contamination/Adulteration

- Requires that food be protected from contamination, obtained from approved food sources and inspected upon receipt.
- Establishes food transportation requirements.
- Requires whole uncut produce to be washed in potable water before use.
- Prohibits use of unapproved additives in food.

Continued---

- Requires use of pasteurized eggs when using raw eggs to make foods such as Caesar salad, hollandaise or Béarnaise sauce, mayonnaise, eggnog, ice cream, and egg fortified beverages.
- Contains provisions for use of molluscan shellfish and game animals for food service.
- Contains criteria for use of reduced oxygen packaging.
- Contains provisions for use of a Hazard Analysis Critical Control Point Plan under certain conditions.
- Contains provisions for issuance of a variance for alternate food safety practices that provide equivalent consumer protection.
- Requires that food facilities be constructed and operated to exclude vermin.
- Excludes live animals, birds or fowl. Exceptions are provided for service animals, dogs under the control of a uniformed law enforcement officer, and aquariums and enclosed aviaries that do not create a public health problem.
- Establishes standards for sanitization and alternate methods of sanitization of food contact surfaces.
- Establishes criteria for dry cleaning methods, use of wiping cloths, clean in place equipment, and use of drying agents.
- Prohibits sulfites on potentially hazardous foods.
- Establishes standards for food storage and display.
- Establishes standards for water potability and temperature.
- Establishes uniform standards for outdoor food service for food facilities that conduct limited food preparation.

Employee Health and Hygiene

- Introduces the concept of identifying a "person in charge" at a retail food facility and specifies the food safety duties and qualifications of said person.
- Establishes requirements for food handler health and hygiene.
- Establishes provisions for use of hand sanitizers.
- Requires that signs be posted for hand washing, no smoking, review of the last inspection report availability, and if no toilet facilities are available to the public if the facility provides table service.

Sanitation

- Restricts sleeping accommodations and sale or service of foods prepared in a private home.
- Contains sanitary provisions for equipment, utensils, linens, facilities, and proper disposal of waste material.

Facilities and Equipment

- Contains provisions for preset tableware.
- Establishes standards for food service equipment.
- Contains provisions for installation and maintenance of plumbing, potable water and waste water tanks, employee changing rooms, hand washing sinks, janitorial sinks, utensil washing sinks, molluscan shellfish tanks, food preparation sinks, and employee and customer toilet facilities.

Continued---

- Contains provisions for ventilation of cooking equipment, comfort, and toilet rooms.
- Requires enclosure of food facilities that conducted extensive food preparation.
- Contains provisions for construction of floors, walls and ceilings of food facilities.
- Establishes uniform requirements for temporary food facilities and mobile food facilities based on type of food preparation and environmental conditions.

FISCAL IMPACT

Potentially significant fiscal impact on DHS and the county health agencies to promulgate regulations and implement the revised retail food facility oversight program.

BACKGROUND AND DISCUSSION

Purpose of the bill

The purpose of this measure is to create some uniformity between California's retail food safety laws and those of other states by modeling California's code after the federal Model Food Code and to base this new code on the best available science.

According to the author, SB 144 represents nearly ten years of work by the California Retail Food Safety Coalition in developing an enhanced food safety law for California that is based on the best available food safety science.

The author believes that ensuring safe food is an important public health priority for our nation and California. Each year in the United States, foodborne illness causes an estimated 76,000,000 illnesses, 324,000 hospitalizations, and 5,000 deaths. The estimated cost of foodborne illness is a staggering $10 - $83 billion annually. Many of these illnesses are preventable by adhering to food sanitation and safety standards.

The author states that child day care facilities, community care facilities, and residential care facilities for the elderly are also exempt from CURFFL with delegation of substandard food safety and sanitation requirements. SB 144 contains language that would remove that exemption. Additionally, CURFFL has become fragmented over time and does not accommodate evolving science and new technologies geared to ensuring a primary public health goal, the safety and security of our food supply.

Arguments in support

SB 144 is sponsored by the California Retail Food Safety Coalition, which is comprised of approximately 65 participants representing industry including restaurants, retailers, hotels and motels, temporary food facilities the local environmental health administrators and the Department of Health Services (DHS).

According to the sponsor, a major step in advancing a science-based food safety system in California has been achieved by the active participation of federal, state, and local regulators and the retail food industry. Representatives from each of these groups have come together in a forum known as the California Retail Food Safety Coalition (CRFSC) with a common goal of protecting and improving public health. Together, using the best available science, the representatives have drafted a proposal for a new food law for

Continued---

California known as the California Retail Food Code (CalCode). The sponsor states that the efforts made by industry and regulators to reach consensus on this new law are commendable and will continue.

The sponsor states that the primary law that regulates the safety of California's retail food service industry, the California Uniform Retail Food Facilities Law (CURFFL), was enacted in 1985 to merge the various overlapping and confusing laws that existed at that time and to provide a body of law that would be applied uniformly throughout all 62 local environmental health jurisdictions in the state. However, in comparison to the best available food safety science found in the United States Food and Drug Administration's (FDA) Model Food Code, CURFFL is lacking critical interventions that scientific studies have shown are more effective in protecting the public's health. According to the sponsor, CURFFL does not accommodate evolving science and new technologies geared to ensuring a primary public health goal: the safety and security of our food supply.

The sponsor argues that CalCode will give California's public, retail food service industry, and state and local health officials and regulators a significantly better body of retail food safety and sanitation law. CalCode does not adopt the Model Food Code verbatim. However, CalCode is clearly based on the FDA Model Food Code and generally adopts its formatting and overall substance; in fact, CalCode is more than 90 percent equivalent to the Model Food Code in terms of its substantive food safety and sanitation content. This fact alone will greatly benefit those businesses that operate in more than one state.

The sponsor states that although a great majority of the Model Food Code is incorporated – either verbatim or in substance – in CalCode, there are some Model Food Code provisions that were intentionally omitted after careful consideration and analysis of the public health implications involved. Where appropriate, a number of CURFFL provisions that are considered to be of importance to the retail food service industry in California have been retained in CalCode.

Further, the entire CRFSC feels very confident that the CalCode represents a comprehensive, visionary, and important step forward in: (1) making sure that the foods and beverages that Californians purchase or consume at retail food establishments are as safe, sanitary, and wholesome; (2) making sure that the manner in which California regulates the retail food service industry is appropriately uniform vis-à-vis with what the other states and the federal government are doing, and providing the optimal efficiency, uniformity, and consistency in the enforcement of the state's retail food service safety and sanitation standards.

Arguments in opposition

The opposition argues that consumer food safety experts did not participate in the review of CURFFL and the drafting of SB 144 and therefore have not been given adequate time to analyze and weigh in on the repeal of CURFFL and the complete recasting of retail food law in California.

Consumer protection organizations are concerned that the coalition that drafted SB 144 did not include any consumer advocacy groups. They believe that they have not had

Continued---

According to HOAC there are many issues of concern that need further review including labeling issues (such as date marking of perishable foods and warnings about uncooked foods), as well as the possible reuse of potentially hazardous food containers.

Comment

Per the direction of the Senate Health Committee, the author's office, proponents and opponents of SB 144 met to discuss the preliminary concerns and amendments to the bill. Some amendments were agreed upon. The author's office agreed to facilitate future meetings to ensure that all policy concerns are addressed. The author has also agreed to bring this bill back to this committee once the bill has been amended.

POSITIONS

Support:
California Retail Food Safety Coalition (sponsor)
California Association of Environmental Health Administrators
California Restaurant Association
California Grocers Association
County of San Diego

Oppose:
California Association of Homes and Services for the Aging *(unless amended*
Consumer Federation of California
Consumers Union
Health Officers Association of California *(unless amended)*
Public Citizen *(unless amended)*

-- END --

Exhibit O

U.S. Food and Drug Administration
Department of Health and Human Services
CENTER FOR FOOD SAFETY AND APPLIED NUTRITION

Food Code

U. S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Food and Drug Administration
College Park, MD 20740

2005

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FDA/Center for Food Safety & Applied Nutrition
Hypertext updated by dms/cjm October 5, 2007

3-6 FOOD IDENTITY, PRESENTATION, AND ON-PREMISES LABELING

Subparts

Accurate Representation

3-601.11 Standards of Identity.

PACKAGED FOOD shall comply with standard of identity requirements in 21 CFR 131-169 and 9 CFR 319 Definitions and standards of identity or composition, and the general requirements in 21 CFR 130 – Food Standards: General and 9 CFR 319 Subpart A – General.

3-601.12 Honestly Presented.

(A) FOOD shall be offered for human consumption in a way that does not mislead or misinform the CONSUMER.

(B) FOOD or COLOR ADDITIVES, colored overwraps, or lights may not be used to misrepresent the true appearance, color, or quality of a FOOD.

Labeling

3-602.11 Food Labels.

(A) FOOD PACKAGED in a FOOD ESTABLISHMENT, shall be labeled as specified in LAW, including 21 CFR 101 - Food labeling, and 9 CFR 317 Labeling, marking devices, and containers.

(B) Label information shall include:

(1) The common name of the FOOD, or absent a common name, an adequately descriptive identity statement;

(2) If made from two or more ingredients, a list of ingredients in descending order of predominance by weight, including a declaration of artificial color or flavor and chemical preservatives, if contained in the FOOD;

(3) An accurate declaration of the quantity of contents;

(4) The name and place of business of the manufacturer, packer, or distributor; and

(5) The name of the FOOD source for each MAJOR FOOD ALLERGEN contained in the FOOD unless the FOOD source is already part of the common or usual name of the respective ingredient (Effective January 1, 2006).

(6) Except as exempted in the Federal Food, Drug, and Cosmetic Act § 403(Q)(3) - (5), nutrition labeling as specified in 21 CFR 101 - Food Labeling and 9 CFR 317 Subpart B Nutrition Labeling.

(7) For any salmonid FISH containing canthaxanthin as a COLOR ADDITIVE, the labeling of the bulk FISH container, including a list of ingredients, displayed on the retail container or by other written means, such as a counter card, that discloses the use of canthaxanthin.

(C) Bulk FOOD that is available for CONSUMER self-dispensing shall be prominently labeled with the following information in plain view of the CONSUMER:

(1) The manufacturer's or processor's label that was provided with the FOOD; or

(2) A card, sign, or other method of notification that includes the information specified under Subparagraphs (B)(1), (2), and (5) of this section.

(D) *Bulk, unPACKAGED FOODS such as bakery products and unPACKAGED FOODS that are portioned to CONSUMER specification need not be labeled if:*

(1) *A health, nutrient content, or other claim is not made;*

(2) *There are no state or local LAWS requiring labeling; and*

(3) *The FOOD is manufactured or prepared on the PREMISES of the FOOD ESTABLISHMENT or at another FOOD ESTABLISHMENT or a FOOD PROCESSING PLANT that is owned by the same PERSON and is regulated by the FOOD regulatory agency that has jurisdiction.*

3-602.12 Other Forms of Information.

(A) If required by LAW, CONSUMER warnings shall be provided.

(B) FOOD ESTABLISHMENT or manufacturers' dating information on FOODS may not be concealed or altered.

Consumer Advisory

3-603.11 Consumption of Animal Foods that are Raw, Undercooked, or Not Otherwise Processed to Eliminate Pathogens.*

(A) Except as specified in ¶ 3-401.11(C) and Subparagraph 3-401.11(D)(3) and under ¶ 3-801.11(D), if an animal FOOD such as beef, EGGS, FISH, lamb, milk, pork, POULTRY, or shellfish is served or sold raw, undercooked, or without otherwise being processed to eliminate pathogens, either in READY-TO-EAT form or as an ingredient in another READY-TO-EAT FOOD, the PERMIT HOLDER shall inform CONSUMERS of the significantly increased RISK of consuming such FOODS by way of a DISCLOSURE and REMINDER, as specified in ¶¶ (B) and (C) of this section using brochures, deli case or menu advisories, label statements, table tents, placards, or other effective written means.

(B) DISCLOSURE shall include:

(1) A description of the animal-derived FOODS, such as “oysters on the half shell (raw oysters),” “raw-EGG Caesar salad,” and “hamburgers (can be cooked to order);” or

(2) Identification of the animal-derived FOODS by asterisking them to a footnote that states that the items are served raw or undercooked, or contain (or may contain) raw or undercooked ingredients.

(C) REMINDER shall include asterisking the animal-derived FOODS requiring DISCLOSURE to a footnote that states:

(1) Regarding the safety of these items, written information is available upon request;

(2) Consuming raw or undercooked MEATS, POULTRY, seafood, shellfish, or EGGS may increase your RISK of foodborne illness; or

Date of Hearing: July 5, 2005

ASSEMBLY COMMITTEE ON HEALTH
Wilma Chan, Chair
SB 144 (Runner) – As Amended: June 28, 2005

SENATE VOTE: 34-1

SUBJECT: Retail food.

SUMMARY: Repeals and reenacts the existing California Uniform Retail Food Facilities Law (CURFFL) as the California Retail Food Code, including substantive changes, effective January 1, 2007. Specifically, some of the substantive provisions of this bill include:

1) General Provisions.

   a) Requires the Department of Health Services (DHS) to provide technical assistance, training, standardization, program evaluation, and other services to local health agencies as necessary to ensure uniform interpretation and application.

   b) Requires DHS to evaluate the program of each local enforcement agency at least once every three years. Requires DHS to prepare a report of the evaluation and program improvements.

2) Definitions.

   a) Defines "permit holder" as an entity that is legally responsible for the operation of the food facility, such as the owner, owner's agent, or other person, and possesses a valid permit to operate a food facility

   b) Defines a "person in charge" as the individual present at a food facility who is responsible for the operation of the food facility.

3) Management and Personnel. Requires the permitholder to be the person in charge or designate a person in charge at the food facility during all hours of operation. Requires the person in charge to ensure that persons unnecessary to the food facility operation are not allowed in the food preparation, storage, or warewashing areas.

4) Employee Health.

   a) Requires a permitholder to require food employee applicants to whom a conditional offer of employment is made and food employees to report to the person in charge, information about their health and health status as they relate to diseases transmissible through food.

   b) Requires a food employee or applicant to report specified information at the outset of jaundice or of an illness as specified.

   c) Lists illnesses diagnosed by a licensed medical practitioner that must be reported. Includes in the list salmonella typhi, salmonella spp, shigella spp, hepatitis A virus.

Includes in the list of symptoms of illnesses that must be reported: diarrhea, fever with abdominal cramps, vomiting, jaundice, sore throat with fever, and lesions.

d) Requires the person in charge to inquire from an applicant or employee whether he or she is subject to one or more high-risk conditions, as specified.

e) Specifies when the person in charge may remove an exclusion or restriction for a food employee diagnosed with an infectious agent, as specified.

f) Requires the person in charge to notify the local health officer when a food employee is diagnosed with an illness, as specified.

g) Establishes provisions for the use of hand sanitizers.

h) Requires signage to be posted for hand washing, no smoking, review of the last inspection report availability, and if no toilet facilities are available to the public, if the facility provides table service.

5) General Food Safety Requirements.

a) Establishes standards for temperature control of potentially hazardous foods.

b) Establishes provisions for using time as a public health control for potentially hazardous foods.

c) Contains exemptions from temperature holding requirements for Korean Rice Cakes and Chinese-style Roast Duck.

d) Clarifies and strengthens provisions for use of molluscan shellfish and game birds.

e) Prescribes that pasteurized eggs can be used in certain uncooked foods.

6) Facilities and Equipment.

a) Contains provisions for preset tableware.

b) Establishes standards for food service equipment.

c) Includes provisions for installation and maintenance of plumbing, potable water, and waste water tanks, employee changing rooms, hand washing sinks, janitorial sinks, utensil washing sinks, food preparation sinks, and employee and customer toilet facilities.

d) Requires enclosure of food facilities that conduct extensive food preparation.

e) Establishes uniform requirements for temporary food facilities and mobile food facilities based on type of food preparation and environmental conditions.

f) Requires that at least five gallons of water be provided exclusively for handwashing for each nonpermanent food facility. Requires that any water need for other purposes be in

addition to the five gallons for handwashing.

g) Requires at least 15 gallons of water to be provided for nonpermanent food facilities that conduct limited food preparation.

7) Physical Facilities.

a) Requires a food facility to be at all times constructed, equipped, maintained, and operated as to prevent the entrance and harborage of animals, birds, vermin, including but not limited to, rodents and insects.

b) Establishes criteria for dry cleaning methods, use of wiping cloths, clean in place equipment and use of drying agents.

8) Permanent Food Facilities. Requires that attachments to walls and ceilings, such as light fixtures, mechanical room ventilation system components, vent covers, wall mounted fans, decorative items, and other attachments to be easily cleanable.

9) Mobile Food Facilities. Establishes uniform requirements for temporary food facilities and mobile food facilities based on type of food preparation and environmental conditions.

10) Nonprofit Charitable Temporary Food Facilities.

a) Requires, except where all food and beverage is prepackaged, hand washing, and utensil washing facilities approved by the enforcement officer, be provided within nonprofit charitable temporary food facilities.

b) Specifies requirements for toilet facilities, food contact surfaces and disposal of liquid waste.

c) Prohibits disposal of potentially hazardous food or beverage stored or prepared in a private home from a nonprofit charitable temporary food facility.

d) Specifies requirements for food and beverages.

e) Allows the presence, in any room where food is served to the public, guests, or patrons, of a guide dog, as defined, accompanied by a totally or partially blind person or deaf person, as specified.

11) Plan Review and Permits.

a) Requires plans for new and remodeled food facilities to be submitted and approved by the local environmental health jurisdiction prior to operation.

b) Requires that a permit be issued for each food facility and has provisions for suspension, revocation, and modification of that permit.

c) Requires registration of supervised care facilities where food service is provided for less than 15 persons.

d) Includes provisions on penalties for operating without a permit.

12) Enforcement and Inspection.

a) Authorizes local jurisdiction to inspect, immediately suspend a permit, conduct hearings, take samples or other evidence, impound food or equipment, and issue reports.

b) Includes provisions for misdemeanor penalties for violations.

c) Makes the owner, manager, or operator of the food facility responsible for violations.

d) Establishes criteria for food facility inspection format, standardization of inspections, and Internet reporting.

13) Child Day Care Facilities, Community Care Facilities and Residential Care Facilities for the Elderly (RCFEs).

a) Includes existing law definition of child day care facilities, community care facilities and RCFEs.

b) Requires the State Department of Social Services (DSS), if and when adequate funding is made available, to develop new regulations regarding food preparation provisions for child day care facilities, community care facilities, and RCFEs to carry out the intent of this section of this bill to ensure the health and safety of individuals.

c) Requires DSS to consult with the State Department of Education in developing proposed food preparation provisions for child day care facilities.

d) Requires DSS to refer repeat and major violations to the appropriate state or local enforcement agency.

e) Requires implementation of provisions imposing additional duties on DHS only when adequate funding is made available.

14) Contains provisions for use of a Hazard Analysis Critical Control Point Plan under certain conditions.

EXISTING LAW:

1) Establishes CURFFL for the regulation of the health and sanitation standards for retail food facilities by DHS and is primarily enforced by local health agencies. CURFFL prohibits a food facility from opening for business without a valid permit issued by the local enforcement agency. Specifies that a violation of any provision of CURFFL is a misdemeanor.

2) Establishes authority of local environmental health jurisdictions to adopt a food safety inspection program with oversight by DHS's Food and Drug Branch.

3) Establishes uniform food safety and sanitation requirements for local jurisdictions to follow. Provisions are also provided to allow a local jurisdiction to adopt a grading system for food facilities, to prohibit any type of food facility, and to regulate the provision of toilet and hand washing facilities.

4) Requires one certified food manager to pass an approved examination for each food facility handling unpackaged potentially hazardous foods with the exception of temporary food facilities and mobile food facilities.

5) Grandfathers local jurisdictions that had a food handler training program in effect since 1998.

6) Requires plans for new and remodeled food facilities to be submitted and approved by the local environmental health jurisdiction prior to operation.

7) Requires that a permit be issued for each food facility and has provisions for suspension, revocation and modification of that permit. Permit fees are established in each local jurisdiction.

8) Provides provisions for penalties for operating without a permit.

9) Provides authority for the local jurisdiction to inspect, immediately suspend a permit, conduct hearings, take samples or other evidence, impound food or equipment, and issue reports.

10) Provides provisions for misdemeanor penalties for violations.

11) Makes the owner, manager, or operator of the food facility responsible for violations.

12) Establishes criteria for food facility inspection format, standardization of inspections, and Internet reporting.

13) Establishes standards for temperature control of potentially hazardous foods. Establishes provisions for using time as a public health control for potentially hazardous foods.

14) Contains exemptions from temperature holding requirements for Korean Rice Cakes and Chinese-style Roast Duck.

15) Establishes cooking standards for certain potentially hazardous foods.

16) Establishes standards for thawing, reheating and cooling of potentially hazardous foods.

17) Requires that food be protected from contamination, obtained from approved food sources and inspected upon receipt.

18) Contains provisions for use of a Hazard Analysis Critical Control Point Plan under certain conditions.

19) Requires that food facilities be constructed and operated to exclude vermin.

20) Excludes live animals, birds or, fowl. Exceptions are provided for service animals, dogs under the control of a uniformed law enforcement officer, aquariums, and enclosed aviaries that do not create a public health problem.

21) Establishes standards for sanitization of food contact surfaces.

22) Prohibits sulfites on potentially hazardous foods.

23) Establishes standards for food storage and display.

24) Establishes standards for water portability and temperature.

25) Establishes requirements for food handler health and hygiene.

26) Restricts sleeping accommodations and sale or service of foods prepared in a private home.

27) Contains sanitary provisions for equipment, utensils. linens, facilities, and proper disposal of waste material.

28) Establishes standards for food service equipment.

29) Contains provisions for installation and maintenance of plumbing, employee changing rooms. hand washing sinks, janitorial sinks. utensils, washing sinks. and employee and customer toilet facilities.

30) Contains provisions for ventilation of cooking equipment, comfort, and toilet rooms.

31) Requires enclosure of food establishments with exceptions for produce stands, swap meet prepackaged food stands, dining areas, open-air barbecues and wood burning ovens, beverage bars, The Mercado La Paloma, and certain outdoor displays and beverage dispensing operations.

32) Contains provisions for construction of floors, walls and ceilings of food facilities.

33) Contains a separate article with separate requirements for open-air barbecue facilities, vending machines, mobile food facilities, mobile food preparation and stationary mobile food preparation units, commissaries, temporary food facilities, nonprofit charitable temporary food facilities, produce stands, certified farmers markets, swap meet prepackaged food stands, satellite food distribution facilities, restricted food service transient occupancy establishments and agricultural home stays, and food facility donations.

34) Exempts child day care facilities, community care facilities and RCFE's from the provisions of CURFFL.

FISCAL EFFECT: According to the Senate Appropriations Committee, DHS has estimated that the costs of implementing this measure would be approximately $1 million for the first year and approximately $1.7 million for each year thereafter. These costs include the costs of promulgating regulations, modifying the regulatory process and enforcement at the state level. Local costs for similar activities related to retail food facilities are indeterminate at this time, but likely to be in excess of $1 million or more per year, and are likely to be offset with higher locally-levied inspection fees.

COMMENTS:

1) PURPOSE OF THIS BILL. According to the author, this bill would create uniformity between California's retail food safety laws and those of other states by modeling California's code after the federal Model Food Code and base this new code on the best available science. The author points out that this bill represents nearly ten years of work by the California Retail Food Safety Coalition, which includes the California Association of Environmental Health Administrators, representatives from the restaurant, retail, hotel, motel, temporary food facilities and DHS. The sponsors point out that this bill continues to ensure that safe food is provided to Californians. Each year in the United States, food borne illnesses causes an estimated 76 million illnesses, 324,000 hospitalizations and 5,000 deaths. Although CURFFL, which was enacted in 1985 to merge the various overlapping and confusing laws that existed at that time and remains the body of law that governs retail food safety in California, there is a need to accommodate evolving science and new technologies to ensure that the safety and security of our food supply. They state that in comparison to the best available safety science found in the United States Food and Drug Administration's (FDA) Model Food Code, CURFFL is lacking critical interventions and that scientific studies have shown that the FDA's Model Food Code is more effective in protecting the public's health.

2) SUPPORT. Supporters, including the California Association of Environmental Health Administrators, California Restaurant Association, and California Grocers Association indicate that this bill promotes uniformity and consistency in retail food safety enforcement; affords greater protection of highly susceptible populations, including children at school and community and elderly care facilities; ensures food safety regulation is based on the best available science; and facilitates understanding and compliance of laws through consolidation of statutory provisions.

3) OPPOSE UNLESS AMENDED. The Health Officers Association of California (HOAC) states it has taken an oppose unless amended position on this bill. HOAC states that there is a lack of directive regarding food establishment employees with gastro-intestinal disorders. HOAC indicates that there should be a clear provision that employees with diarrhea, nausea, or vomiting be prohibited from working with food. However, it appears that this concern has been addressed by recent amendments to this bill. In addition, HOAC believes a section is needed regarding reporting employee illnesses to a local authority. Food establishments should report any food safety problems to inspectors, rather than waiting for inspectors to raise the issue. Finally, HOAC states that there are many other issues it is concerned about and needs to research further. These issues include labeling and possible reuse of potentially hazardous food containers.

4) OTHER OPPOSITION. The committee did not receive any letter of opposition from these organizations but according to the Senate Health Analysis, the California Association of

Homes and Services for the Aging, Consumer Federation of California and Consumers Union have an oppose or oppose unless amended positions on this bill. The Senate Health Committee analysis states that these organizations argue that consumer food safety experts did not participate in the review of CURFFL and the drafting of this bill and were not given adequate time to analyze and weigh in on the repeal of CURFFL and the complete recasting of retail food law in California. Furthermore, consumer protection organizations are concerned that the coalition that drafted this bill did not include any consumer advocacy groups. They also indicate they have not had adequate time to review this legislation and compare it to CURFFL or the FDA's Model Food Code.

5) POLICY QUESTIONS. The bill on page 138, lines 19-23, would require DSS to refer repeat and major violations to the appropriate state or local enforcement agency when its inspections of child day care facilities, community care facilities and RCFEs reveal violations. What constitutes major violations? Are DSS inspectors sufficiently trained and familiar with this bill to ensure they refer the appropriate violations?

6) COMMENTS. This bill proposes a significant restructuring of the statute as well as multiple, substantive changes to California's food safety requirements. However, these major changes are proposed even though this bill was amended within a few days of the deadline for this committee to complete the analysis. The sponsors were unable to provide a detailed comparison of existing law and the proposed changes in a timeframe to permit a thorough review and analysis.

REGISTERED SUPPORT / OPPOSITION:

Support

California Association of Environmental Health Administrators (co-sponsor)
California Grocers Association
California Restaurant Association
County of San Diego

Opposition

None on file.

Analysis Prepared by: Rosielyn Pulmano / HEALTH / (916) 319-2097

FILE NO. 080826 ORDINANCE NO. 195-08

[Menu Labeling at Chain Restaurants.]

Ordinance amending Sections 468.2, 468.3, 468.4, 468.5, 468.6, and 468.8 of the San Francisco Health Code to (1) set rounding rules for disclosure of nutritional information, (2) allow nutritional information for pizzas to be listed per serving size, (3) allow chain restaurants to display the information required by Section 468.4 in brochures and booklets, (4) authorize the Director of the Department of Public Health to adopt rules and regulations to enforce these provisions, and (5) extend the deadlines for electronic reporting and disclosure on menus, food tags, and posters.

Note: Additions are *single-underline italics Times New Roman*; deletions are ~~*strikethrough italics Times New Roman*~~. Board amendment additions are double underlined. Board amendment deletions are ~~strikethrough normal~~.

Be it ordained by the People of the City and County of San Francisco:

Section 1. The San Francisco Health Code is hereby amended by amending Sections 468.2, 468.3, 468.4, 468.5, 468.6, and 468.8 to read as follows:

SEC. 468.2. DEFINITIONS.

(a) "Chain Restaurant" means a Restaurant within the City and County of San Francisco that offers for sale substantially the same Menu Items, in servings that are standardized for portion size and content, and is one of a group of 20 or more Restaurants in California that either: (1) operate under common ownership or control; or (2) operate as franchised outlets of a parent company, or (3) do business under the same name.

(b) "Director" shall mean the Director of Health, or his designated agents or representatives.

(c) "Food" means any substance in whatever form for sale in whole or in any part for human consumption such as, for example, meals, snacks, desserts, and beverages of all kinds.

(d) "Food Tag" shall mean a label or tag that identifies any Food item offered for sale at a Chain Restaurant, such as, for example, a label placed next to a cherry pie showing a picture of a cherry and listing the price per slice.

(e) "Menu" means any list *or pictorial display* of Food, *and price(s),* offered for sale at a Restaurant including menus distributed or provided outside of the Restaurant, but does not include a Menu Board.

(f) "Menu Board" means any list or pictorial display of Food, *and price(s),* offered for sale at a Restaurant that is posted in a Restaurant and intended for shared viewing by multiple customers such as, for example, back-lit marquee signs above the point of sale at fast-food outlets and chalk boards listing offered Food items or any list of Food offered for sale at a Restaurant that is posted and intended for viewing by customers purchasing Food to go, such as, for example, a drive-through menu.

(g) "Menu Item" means an item described on a Menu, a Menu Board, or a Food Tag that is prepared, un-prepackaged Food; and also means a combination item appearing on a Menu, a Menu Board, or a Food Tag such as, for example, a "kids meal," that contains any prepared, un-prepackaged Food, such as a hamburger, and any prepackaged Food, such as a carton of milk.

(h) "Restaurant" means a facility at which any prepared, un-prepackaged Foods are offered for sale and consumption on or off the premises such as, for example sit-down restaurants; cafes; coffee stands; and fast-food outlets, but not grocery stores. "Restaurant"

may also include separately owned food facilities that are located in a grocery store but does not include the grocery store.

SEC. 468.3. MENU LABELING REQUIRED AT CHAIN RESTAURANTS.

(a) Required Nutritional Information. Except as provided in Subsection (h), each Chain Restaurant shall make nutritional information available to consumers for all Menu Items. This information shall include, but not be limited to, all of the following, per Menu Item, as usually prepared and offered for sale:

(1) Total number of calories, *expressed to the nearest 10 calories for values above 50 calories and to the nearest 5 calories for values 50 calories and below*;

(2) Total number of grams of saturated fat, *expressed to the nearest gram for values 5 grams and above, to the nearest half gram for values below 5 grams, and as 0 for values below one half gram*;

(3) Total number of grams of carbohydrates, *expressed to the nearest gram for values 1 gram and above, as "less than 1g" for values below 1 gram, and as 0 for values below one half gram*; and

(4) Total number of milligrams of sodium, *expressed to the nearest 10 milligrams for values above 140 milligrams, to the nearest 5 milligrams for values between 5 and 140 milligrams, and as 0 for values below 5 milligrams*.

(b) Information on Menus.

(1) Each Chain Restaurant that uses a Menu shall provide the nutritional information required by ~~s~~Subsection (a) next to or beneath each Menu Item using a size and typeface that is clear and conspicuous.

(2) Each Chain Restaurant that uses a Menu shall include the following statement on the Menu in a clear and conspicuous manner: "Recommended limits for a 2,000 calorie daily diet are 20 grams of saturated fat and 2,~~3~~400 milligrams of sodium."

(c) Information on Menu Boards.

(1) Each Chain Restaurant that uses a Menu Board shall provide on the Menu Board the nutritional information required by Subsection (a)(1) next to or beneath each Menu Item on the Menu Board using a font and format that is at least as prominent, in size and appearance, as that used to post either the name or price of the Menu Item.

(d) Information on Food Tags. Each Chain Restaurant that uses a Food Tag shall provide on the Food Tag the nutritional information required by *s*Subsection (a)(1) using a font and format that is at least as prominent, in size and appearance, as that used to post either the name or price of the Menu Item.

(e) Range of nutritional information for different flavors and varieties.

(i) If a Chain Restaurant offers a Menu Item in more than one flavor or variety (such as beverages, ice cream~~, *pizza*~~, or doughnuts) and lists the item as a single Menu Item, the range of values for the nutritional information for all flavors and varieties of that item (i.e., the minimum to maximum numbers of calories) shall be listed for each size offered for sale. *If the Menu Item's flavors and/or varieties include diet items containing less than 10 calories, the Chain Restaurant shall post a separate range of values for such diet items.*

(ii) If a Chain Restaurant offers pizzas in different sizes as Menu Items, the nutritional information required by Subsection (a) may be listed on the Menu and/or Menu Board by serving size for each type of pizza if the Chain Restaurant does both of the following: (1) displays the definition of the serving size (i.e., 1 slice) on the Menu and/or Menu Board using a size and typeface that is clear and conspicuous, and (2) displays the number of servings for each size of each type of pizza next to or

beneath the price for each size using a font and format that is at least as prominent, in size and appearance, as that used to post either the name or price of the Menu Item.

(f) Disclaimers. Menus, Menu Boards, and Food Tags may include a disclaimer that indicates that there may be minimal variations in nutritional content across servings, based on slight variations in overall size and quantities of ingredients, and based on special ordering.

(g) Verifiable and Reliable Information Required.

(1) The nutrition information required by this section and Section 468.4 shall be based on a verifiable analysis of the Menu Item, which may include the use of nutrient databases, cookbooks, laboratory testing, or other reliable methods of analysis.

(2) A Restaurant is in violation of this section and Section 478.4 if the provided nutritional information required by these sections:

(i) Is not present in the location or in the form required by these sections:

(ii) Is different than what the Restaurant knows or believes to be the true and accurate information; or

(iii) Deviates from what actual analysis or other reliable evidence shows to be the average content of a representative sample of the Menu Item by more than 20%.

(h) Food Items Excluded. This section and ~~s~~Section 468.4 shall not apply to:

(1) Items placed on the table or at a counter for general use without charge, such as, for example, condiments:

(2) Alcoholic beverages; and

(3) Items that are on the Menu, Menu Board or Food Tag for less than 30 *consecutive* days in a calendar year.

SEC. 468.4. NUTRITION INFORMATION REQUIRED TO BE DISCLOSED ON DISCLOSURE MEDIA OTHER THAN MENUS, MENU BOARDS AND FOOD TAGS.

(a) Each Chain Restaurant shall make the following nutrition information available to consumers per Menu Item, as usually prepared and offered for sale, on the disclosure media provided for in ~~s~~Subsection (c): calories, protein, carbohydrates, total fat, saturated fat, artificial trans fat, cholesterol, fiber and sodium. The nutrition information shall consist of the following items:

(1) A heading titled "Nutrition Information" or equivalent heading acceptable to the Department of Public Health.

(2) The nutritional information required by Section 468.3(a).

(3) Protein~~, fiber, total fat, and trans fat~~ shall be expressed to the nearest gram *for values 1 gram and above, as "less than 1g" for values below 1 gram, and as 0 for values below one half gram* ~~per serving~~.

(4) Cholesterol shall be expressed to the nearest 5 milligram*s for values above 5 milligrams, expressed as "less than 5 mg" for values between 2 and 5 milligrams, and expressed as zero for values below 2 milligrams* ~~per serving~~.

(5) Fiber shall be expressed to the nearest gram for values 1 gram and above, as "less than 1g" for values below 1 gram, and as 0 for values below one half gram

(6) Total fat and artificial *trans fat shall be expressed to the nearest gram for values 5 grams and above, to the nearest half gram for values below 5 grams, and as 0 for values below one half gram.*

(b) Customers must be able to obtain nutrition information without the necessity of purchasing food.

(c) The information required by ~~s~~Subsection (a) must be disclosed, in a size and typeface that is clear and conspicuous, *either* on a printed poster of a size no smaller than ~~eighteen (~~18~~)~~ inches by ~~twenty four (~~24~~)~~ inches *or in a printed brochure or booklet, that is* displayed in a conspicuous place and readily visible to customers either: (1) at the point of

sale, (2) near the front door; (3) on or near the host/hostess desk or reception area, or (4) at any point in or near the entryway or waiting area of the restaurant.

SEC. 468.5. REPORTING REQUIREMENTS.

By ~~*July*~~ *August* 1, 2008, and July 1st of every year thereafter, Chain Restaurants shall report to the Department of Public Health the information required by Sections 468.3 and 468.4 in an electronic format determined by the Department. The Department shall make this information available to the public.

SEC. 468.6. PENALTIES AND ENFORCEMENT.

(a) Cumulative Remedies. The remedies provided by this section are cumulative and in addition to any other remedies available at law or in equity.

(b) Administrative Remedies. The Director may enforce the provisions of Sections 468.3 through 468.5 by serving a Notice of Violation requesting a Chain Restaurant to appear at an administrative hearing before the Director at least 20 days after the Notice of Violation is mailed. At the hearing, the Chain Restaurant cited with violating the provisions of these sections shall be provided an opportunity to refute all evidence against it. The Director shall oversee the hearing and issue a ruling within 20 days of its conclusion. The Director's ruling shall be final. If the Director finds that a Chain Restaurant has violated any of the provisions of Sections 468.3 through 468.5 or refuses to comply with these sections, the Director may order either of the following penalties:

(1) Suspension or revocation of the permit issued by the Director pursuant to Sections 451 et seq. of this Code; or

(2) An administrative fine in an amount (1) not exceeding one hundred dollars ($100.00) for a first violation; (2) not exceeding two hundred dollars ($200.00) for a second violation within one year; (3) not exceeding five hundred dollars ($500.00) for each additional

violation within one year. In assessing the amount of the administrative penalty, the Director shall consider any one or more of the relevant circumstances presented by any of the parties to the hearing, including but not limited to the following: the nature and seriousness of the misconduct, the number of violations, the persistence of the misconduct, the length of time over which the misconduct occurred, the willfulness of the Chain Restaurant's misconduct, and the Chain Restaurant's assets, liabilities, and net worth. Any penalty assessed and recovered pursuant to this paragraph shall be paid to the City Treasurer and credited to the Department Environmental Health Section Special Revenue Account.

(c) Civil Penalties. Violations of Sections 468.3 through 468.5 are subject to a civil action brought by the City Attorney, punishable by a civil fine not less than two hundred fifty dollars ($250.00) and not exceeding five hundred ($500.00) per violation. Unless otherwise specified in this section, each day of a continuing violation shall constitute a separate violation. In assessing the amount of the civil penalty, the court shall consider any one or more of the relevant circumstances presented by any of the parties to the case, including but not limited to the following: the nature and seriousness of the misconduct, the number of violations, the persistence of the misconduct, the length of time over which the misconduct occurred, the willfulness of the defendant's misconduct, and the defendant's assets, liabilities, and net worth. Any penalty assessed and recovered in an action brought pursuant to this paragraph shall be paid to the City Treasurer and credited to the Department Environmental Health Section Special Revenue Account.

(d) Action for Injunction. The City Attorney may bring a civil action to enjoin a violation of Sections 468.3 through 468.5.

(e) Aiding and Abetting. Causing, permitting, aiding, abetting, or concealing a violation of any provision of Sections 468.3 through 468.5 shall also constitute a violation of this ordinance.

(f) Enforcement Agency. The Department of Public Health shall supervise compliance with Sections 468.3 through 468.5 and shall enforce those sections. *The Director may adopt rules and regulations to give effect to those sections. In addition to the reporting requirements of Section 468.5, the Director may request information from Chain Restaurants that is reasonably necessary to the enforcement of those sections* ~~including, but not limited to, recipe information, provided however that the Department shall keep any proprietary information confidential to the maximum extent permitted by applicable laws~~. *The Department may impose the penalties provided in this section for failure to comply with such a request.* Notwithstanding, any other person legally permitted under federal law, under state law, under Sections 468.3 through 468.5, or under other provisions of this Code to enforce a provision of these sections may enforce that provision. Such persons may include, for example: peace officers; code enforcement officials; and City officials, employees, and agents.

(g) Fees. In order to implement the requirements set forth in this ordinance, the Department of Public Health is hereby authorized to impose a surcharge of $350.00 for the permit issued to Chain Restaurants pursuant to Sections 451 et seq. of this Code.

SEC. 468.8. OPERATIVE DATE.

The disclosure requirements set forth in Sections 468.3 ~~*(b) (Menus), 468.3(d) (Food Tags),*~~ and 468.4(c)~~*, shall become operative ninety (90) days after the effective date of this ordinance. The disclosure requirements set forth in Section 468.3(c) (Menu Boards)*~~ shall become operative *on September 20, 2008.* ~~*one hundred and fifty (150) days after its effective date.*~~

APPROVED AS TO FORM:
DENNIS J. HERRERA, City Attorney

By: _____
FRANCESCA GESSNER
Deputy City Attorney

City and County of San Francisco

City Hall
1 Dr. Carlton B. Goodlett Place
San Francisco, CA 94102-4689

Tails

Ordinance

File Number: 080826 **Date Passed:**

Ordinance amending Sections 468.2, 468.3, 468.4, 468.5, 468.6, and 468.8 of the San Francisco Health Code to (1) set rounding rules for disclosure of nutritional information, (2) allow nutritional information for pizzas to be listed per serving size, (3) allow chain restaurants to display the information required by Section 468.4 in brochures and booklets, (4) authorize the Director of the Department of Public Health to adopt rules and regulations to enforce these provisions, and (5) extend the deadlines for electronic reporting and disclosure on menus, food tags, and posters.

July 29, 2008 Board of Supervisors — PASSED ON FIRST READING

Ayes: 11 - Alioto-Pier, Ammiano, Chu, Daly, Dufty, Elsbernd, Maxwell, McGoldrick, Mirkarimi, Peskin, Sandoval

August 5, 2008 Board of Supervisors — FINALLY PASSED

Ayes: 11 - Alioto-Pier, Ammiano, Chu, Daly, Dufty, Elsbernd, Maxwell, McGoldrick, Mirkarimi, Peskin, Sandoval

File No. 080826

I hereby certify that the foregoing Ordinance was FINALLY PASSED on August 5, 2008 by the Board of Supervisors of the City and County of San Francisco.

Angela Calvillo
Clerk of the Board

8-7-08
Date Approved

Mayor Gavin Newsom